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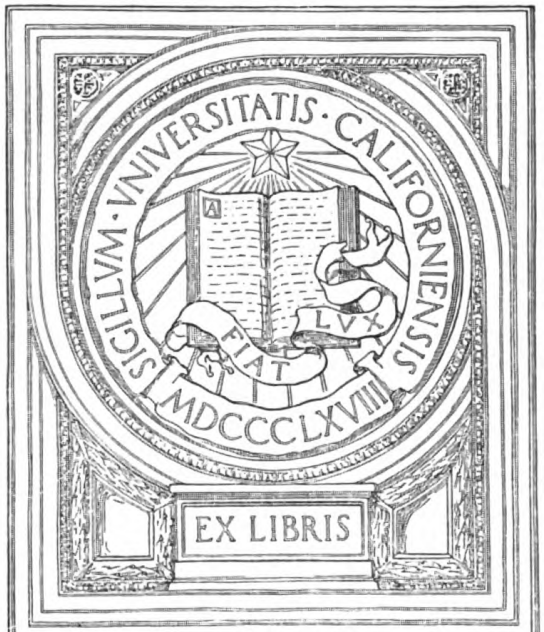

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We regret that owing to the war the number of pages must be diminished for a time.

ELECTRICAL TREATMENT OF PARALYSIS.*

By E. P. CUMBERBATCH, B.M.Oxon., M.R.C.P.,

Medical Officer in charge of the Electrical Department, St. Bartholomew's Hospital.

IN this lecture I wish to describe the electrical treatment of paralysis by modern methods. Little or no information is to be found on this subject in books on medicine and surgery. Yet cases of paralysis are seen often enough in general practice, and much could be done in the way of electrical treatment by the practitioner himself, or under his instruction and supervision by a nurse, or even by one of the patient's friends. Amongst the poor in hospital practice are seen the good effects of correctly applied electrical treatment, and it is the failure to adopt the right methods of carrying it out that has been responsible for the scepticism prevalent in some quarters concerning this form of treatment. In the absence of a more elaborate outfit, much can be done with simple and inexpensive apparatus.

In a case of paralysis the electrical treatment should not be confined solely to the paralysed muscles, but should include the damaged nerves, as far as is possible, and the cause that is responsible for the paralysis.

A simple outfit for the home treatment of paralysis should include: (1) a source of electrical current; (2) suitable electrodes for leading the current into the body.

The current to be used.—The first point to be decided is, whether the current from the coil (faradic current) should be used, or the current from a battery of cells (galvanic current). It is commonly taught that if the muscles show the reaction of degeneration the galvanic current should be used,

* Lecture delivered at the Polyclinic.

and that if they show reactions of the normal type the faradic current should be applied. This teaching is based rather on theoretical grounds than on clinical and experimental observations, and each current has at different times been regarded as superior to the other. It is, therefore, not likely that one is much more efficient than the other for the treatment of the paralysed muscles.

In the Electrical Department at St. Bartholomew's Hospital the electrical currents for the treatment of paralysis are taken from the mains. As such, they would be far too strong to pass through the body, and they must therefore be reduced by suitable resistances. Now if the direct current (called also the "continuous" or "galvanic" current) were used the patient might, through accidental short-circuiting, receive the full strength of the mains' current; and, if he were in a full-length bath, the result would be disastrous. On the other hand, if the alternating current is used, it need not be sent directly through the baths, but through the primary of an induction coil. This current, alternating to-and-fro in the primary, induces currents in the secondary coil, and these currents can be sent through the bath. In this way the mains circuit and the circuit including the bath are independent, and not in metallic connection with each other, as the primary and secondary coils are completely insulated from each other. On account of the risk that attends the use of the galvanic current from the mains, this current is not supplied to the baths in St. Bartholomew's Hospital. The patient receives instead the induction current from the coil, whether his muscles show the reaction of degeneration or not. Recovery of power and return of the normal reactions are frequently seen.

For home treatment of paralysed muscles the current may be taken from an induction coil. The coil can be worked from a dry cell. The selection of the coil is a matter of importance. There are many patterns on the market; some are expensive and needlessly elaborate. The coil chosen should be one that produces the strongest muscular contractions with the least amount of discomfort. Such a coil is that made by Schall and originally designed by Dr. Lewis Jones. It is enclosed in a portable case with a dry cell and a drawer for cables and accessories.

The electrodes.—By "electrode" is meant the conductor which is placed on the skin, so as to lead the current into the underlying tissues. The electrodes must be of large size, so as to cover a large area of skin. The small button-size electrodes are quite inefficient. The larger the electrodes the less the discomfort, and the greater the current that can be sent through the body. The strength of current that can be tolerated depends upon the degree of stimulation of the sensory nerve-endings in the skin and the force of the contraction of the underlying muscles. If a small electrode is used the current is concentrated at its point of entry, producing strong stimulation of a small area of skin, and forcible contraction of the muscle in the immediate neighbourhood. Having passed through the skin the lines of flow of the current diverge, and so the more distant muscles receive very little current. By using an electrode that completely covers the part receiving treatment, the area of entry of the current through the skin is greatly increased, so that considerably more current can pass into the tissues without discomfort, while the muscles also receive considerably more current, and with more even distribution.

The electrode may be made of lint, about six layers in thickness (twelve layers should be used if the galvanic current is to be applied). A folded towel may be used instead of the lint. The electrodes should be thoroughly moistened with warm water to which a small quantity of salt has been added, and placed over the parts to be treated. A metal plate smaller than the electrode, with a terminal on one side, and a layer of felt covering the other side, is moistened with the salt solution, and placed on the electrode and connected with the coil.

If the forearm and hand are to be treated, they may be immersed in water in an arm bath, such as that fitted to a Schnee bath, or one of approximately the same shape and made of non-conducting material. A metal plate may be immersed at each end, and the current sent through the water from end-to-end. The forearm and hand are then traversed by the current, and the water acts as an electrode of the largest possible size. Both forearms, if need be, can be immersed simultaneously. For the treatment of infantile paralysis of the lower limbs in children, a similar arrangement can be used and will be described later.

How the current is applied.—The regulation of the strength of the current is a matter of much

importance. The current should be made to vary continuously in strength between zero and the maximum that the patient can bear. Currents of strength unvaried are much less effective, and may do actual harm to the muscle. All the baths at St. Bartholomew's are supplied with currents that rhythmically vary their strength. There are various mechanical instruments for producing such variation. They are somewhat expensive. A method of obtaining rhythmic variations without such instruments, and suitable for home application, will now be described. When both electrodes are in position and connected to the coil, the latter is put in action. The current now passes through the body, and its strength can be varied rhythmically by sliding the secondary coil over the primary. If the secondary is permanently fixed over the primary, the regulating device is a metal sleeve which can be pulled over the iron core. Rhythmic variation can then be effected by moving the sleeve to-and-fro over the core. A point should be marked (on the metal sleeve or on the frame along which the secondary slides over the primary) at which the maximum bearable strength of current is given, so that the operator can avoid passing beyond it. Another point might be marked at which the current just fails to produce muscular contraction. The current can now be made to wax and wane between zero and maximum, reaching maximum in about two seconds and zero again in the same time.

In some cases, the patient might be able to do the necessary movements himself, and produce the rhythmic variation unaided.

Another way of producing variation of the current strength is to replace the electrode that covers the paralysed muscles by a well-padded metal disc with a handle attached. The disc, *which should be at least three inches in diameter*, is connected by a cable to one of the terminals of the coil, and, after moistening with warm salt solution, is stroked along the skin over the paralysed muscles from origin to insertion, lifted off and again stroked in the same direction. In this way variation of the current strength is effected.

If the paralysed muscles belong to a limb, with an antagonistic group which is not paralysed, the limb should be supported by a splint to prevent the healthy muscles, which will vigorously contract when the current passes through them, pulling on and stretching the paralysed muscles. The support should be worn until the latter have recovered.

Treatment of the affected nerves.—The treatment of the paralysed muscles should at the same time include the nerves that have been damaged. The treatment that has just been described for the muscles will be suitable for the affected nerves. The parts of the latter that lie between and within the muscles will share the treatment that is given to the latter. The indifferent electrode may be placed over the main nerve trunk from which the paralysed muscles have drawn their supply. If the paralysis has a spinal origin the indifferent electrode should be placed over the vertebral column.

If the nerves have been compressed or otherwise mechanically injured, the *galvanic* current would probably be more effective at the commencement of the treatment. Rhythmic variation can be effected by means of the movable electrode already described. The latter can be stroked along the paralysed muscles. If the nerves are in a condition of inflammation, the galvanic current again would probably be more effective at the commencement of the treatment till the inflammation had subsided.

When the galvanic current is applied it may be used at the same time for the introduction of ions into the affected nerves. This may be done with advantage when pain accompanies the paralysis. The electrode covering the painful area should be soaked in one per cent. sodium salicylate solution and should be connected with the negative pole. The other electrode, if it also covers a painful area, might be soaked in a one per cent. solution of quinine hydro-chloride. The quinine ions enter from the pad connected to the positive pole.

It is possible that quinine ions and salicylate ions have some beneficial influence on inflammatory processes in nerves, even when no pain is present.

If no galvanic current is available the faradic current should be used from the commencement, although, of course, the latter cannot bring about the entry of ions. If the contraction of muscles causes pain, the faradic current should not be used, and if the galvanic current is used it should not vary its strength; otherwise contractions will occur. It should be used for the introduction of ions till the pain has disappeared, and then the treatment of the paralysed muscles can be commenced.

The galvanic current may be taken from a

portable box containing a battery of thirty-two dry cells. They can be obtained from any instrument-maker. The cells will last as long as a year before a refill is required.

With regard to the *cause* responsible for the paralysis much cannot be done by electrical methods. In many cases, of course, the cause is no longer operative when the patient comes for treatment, and then we have to treat only its effects. When the paralysis is the result of unknown toxic agents, treatment of the muscles by rhythmic currents will cause contraction, and this will aid elimination of the poisons from the limbs. In cases in which the cause is unknown and progressive the most that can be expected from electrical treatment is some temporary improvement of the condition of the muscles.

The effects of electrical treatment of paralysed muscles and nerves.—The effects of electrical applications on paralysed parts may be briefly considered. The greater quantity of blood that passes to a limb is required for the muscles. A limb in which the muscles are actively contracting receives a much larger quantity of blood than when they remain in a relaxed condition.

But when a muscle is relaxed it is not really at rest, for there is still some continuous contraction or tone, and chemical changes and development of heat are still taking place. A muscle that is in a condition of flaccid paralysis is a toneless muscle and ceases work entirely. The blood supply to the muscle becomes exceedingly small, food supply diminishing, waste products (and probably toxins) accumulating. The muscle hangs loosely from its extremities. If it persists in this condition the fibres degenerate and are replaced by connective tissue. The other tissues of the limb, if many muscles are paralysed, show the result of impoverished blood supply. The skin becomes cold and blue, and chilblains and ulcers develop.

If, now, rhythmic applications of electrical currents are made, all the tissues, deep and superficial, are stimulated. The blood supply to the limb increases, and its temperature rises. The muscles are stimulated, even if they show the reaction of degeneration and do not actually contract.

An increased production of heat and an increased respiratory exchange follows the stimulation of muscles, even when so gentle that no contraction is produced. This was shown by D'Arsonval. If many of the muscles of a limb are paralysed the other tissues, which suffer also from the insufficient blood supply, benefit from the general stimulation and improvement of the circulation following the electrical application.

Some examples will now be given so as to show how the principles laid down may be applied when treating paralysis in different regions.

In the forearm the extensors are frequently paralysed, resulting in "wrist-drop." The cause may be pressure on the musculo-spiral nerve by a crutch or the back of a chair. One electrode, in the form of a moistened pad, should be applied to the part where the pressure was exerted. The other electrode should be applied to the extensor aspect of the forearm so as to cover the paralysed muscles. This electrode may be fixed or movable according to the method available for obtaining rhythmic variation of the current, as described above. To prevent *active* stretching of the paralysed extensors by their healthy antagonists during the treatment, the forearm should be laid, palmar surface down, on a flat surface. A splint should be worn in the intervals between the sessions of treatment so as to prevent *passive* stretching of the paralysed muscles.

If the paralysis is due to some other cause such as a neuritis of the nerve trunks or brachial plexus, the affected nerve should in the same way be included in the treatment by placing one of the electrodes over them.

Cases of facial paralysis (Bell's palsy), if seen in the early stages, do best, in the writer's experience, if treatment is applied to the facial nerve. The active electrode, in the form of a padded disc as described above, should be soaked in sodium salicylate solution and stroked over the face from the region of the stylo mastoid foramen along the branches of the nerve. In this way the inflamed nerve trunk is ionised and, in addition, the paralysed muscles are stimulated.

Children, with infantile paralysis of their lower limbs, can be treated in a small bath made of china or other non-conducting material. The child sits in the bath in warm water that covers his lower limbs and lumbar region of his spinal cord. The faradic current is directed through the water by means of two metal plates hung over the ends of the bath and immersed in the water. Rhythmic variation can be produced in the way described. By this method a large current can be borne by the child without pain. Blueness of the skin and chilblains, if present; will disappear quickly under this treatment, and the nutrition of the weakened but surviving muscles and the cells of origin of their nerves is maintained.

January 4th, 1915.

IRITIS.*

By LIONEL J. PISANI, F.R.C.S.,
Ophthalmic Surgeon to the Metropolitan Hospital.

In endeavouring to find a subject which would be of interest to you gentlemen who are in general practice, it has appeared to me that I could not do better than devote this lecture to iritis, its causes, symptoms, diagnosis and treatment. I propose to exclude iritis of traumatic origin, and also iritis arising from extension of disease from neighbouring structures, such as the cornea, sclera, or from the interior of the eye. I will deal only with "endogenous" iritis, that is iritis in which the infective agent is carried through the circulation.

Ætiology.

The chief causes of endogenous iritis are syphilis, gonorrhœa, tubercle, infection from a septic focus elsewhere, and acute infectious diseases.

I may mention rheumatism which, until recently, was considered to be one of the most common causes of iritis, not because of the association of rheumatic fever with iritis, but because a history of muscular or arthritic pains could be so readily obtained. Pains of this character are frequent in syphilitic, gonorrhœal, or septic infections, and recent experiments of Rosenow,† of Chicago, have shown that these pains are due to bacteria carried by the blood lodging in the capillaries of the muscles and fibrous tissues. Here the bacteria instead of multiplying and setting up a focus of suppuration, perish, liberating toxins which cause destruction of the surrounding cells and their subsequent replacement by fibrous tissue, producing what has been termed "myositis" or "fibrositis."

I may mention that Paynton and Paine‡ have by injecting intravenously the *Diplococcus rheumaticus* in rabbits, produced acute rheumatism, and in two cases associated with iritis, but this does not help us very much because as you know iritis is a rare complication of rheumatic fever. Moreover, the diplococcus is not generally accepted as the cause of rheumatism.

It has hitherto been considered doubtful whether iritis was due to the conveyance of bacteria to the iris or to toxins circulating in the blood. It has been difficult to demonstrate bacteria in the delicate iris tissue, but the *Spirochæta pallida* and the gonococcus have been found in the aqueous in cases of iritis. It seems to me that the experimental evidence which I have mentioned affords an explanation as to the mode of infection in such a vascular structure as the uvea.

I need not deal further with syphilis, gonorrhœa and tubercle in which the infective agent is well known. In iritis arising from septic areas in other parts of the body we have to deal with a bacterial (staphylococcal or other) infection of the blood. Iritis has been found associated with pyorrhœa alveolaris, or other septic diseases of the mouth or throat, naso-pharynx, sinuses, uterine cavity and appendages, furuncles, boils, etc.

As you know, gentlemen, not very long ago there was a very interesting discussion at the Royal Society of Medicine on intestinal toxæmia. Among many other diseases attributed to absorption of toxins from the bowels iritis and other diseases of the uveal tract were mentioned. Quite recently Prof. Adami, of Montreal,* has shown that the toxins generated in the bowels cannot be considered to be the cause of disease, but that there is an infection of the blood by the passage of bacteria from the bowel into the circulation. The bowel thus acts as a source of infection in the same way as any other septic focus.

Cases of iritis arising in the course of acute infectious diseases do not often come to our notice, but it may be mentioned that iritis is most commonly found in relapsing fever, a disease in which the blood swarms with the spirochæte, or *Spirillum Obermeiri*, a spirochæte which is similar to the *Spirochæta pallida*, the infective agent of syphilis.

More recently another blood organism has been shown to cause inflammation of the uvea, the *Trypanosoma Gambiense*, the parasite of sleeping sickness.

Symptoms.

What the Patient complains of.

(1) *Pain* first in the eyeball, after, in the course of

* Post-Graduate Lecture at the Metropolitan Hospital.

† Rosenow, 'Journ. Amer. Med. Assoc.,' lxi, 1913, p. 2007.

‡ 'Trans. Ophth. Soc. U.K.,' xxiii, p. 40.

* 'Brit. Med. Journ.,' January 24th, 1914, p. 177.

distribution of the supra-orbital nerve, occasionally extending to the other branches of the fifth nerve below the orbit. The pain is severe, of a stabbing character, and worse at night.

(2) *Disturbance of vision* due to turbidity of the aqueous, or effusion of lymph into the pupillary area.

(3) *Photophobia* and (4) *Lacrymation.*—These two are not marked except in severe cases.

What you see in Iritis.

(1) *Ciliary injection.*—A zone of diffused, pale red or pale violet colour, surrounding the cornea, due to congestion of the anterior ciliary vessels. Ciliary injection is common to all diseases of the cornea, iris or ciliary body.

(2) *Changes in the colour of the iris.*—At first there is hyperæmia in which the individual vessels can be seen, later with increased exudation a blue iris becomes greenish, a brown iris reddish brown.

(3) *Exudates from the inflamed iris* into its substance, into the pupillary area, or behind the iris producing synechiæ.

(4) *Changes in the character of the aqueous*, such as turbidity with alteration in its composition, the presence of exudates; occasionally blood may be found in the anterior chamber.

Syphilitic iritis appears at any time between the second and ninth month after infection, sometimes as late as the eighteenth month. The iritis first affects one eye then the other. Occasionally it appears as a tertiary manifestation. It is frequently seen at the time that the secondary eruptions appear on the skin, it sometimes assumes a nodular or papular form, the so-called "condyloma of the iris." The nodules are of a yellowish-red colour, varying in size from a hemp seed to that of a small pea and are situated either at the ciliary or pupillary margins, they readily disappear under treatment, leaving well-marked synechiæ.

Gonorrhœal iritis is the result of systemic infection by the gonococcus; an attack of arthritis intervenes between infection and the starting of the iritis, but ocular manifestations may precede the arithitis. Of all the forms of iritis it is perhaps the one which is the most subject to recurrences. For a long time it was difficult to explain these recurrences so long after infection. Some years ago the existence of posterior synechiæ were believed to be the cause, but when it was found that cases with synechiæ resulting from traumatic iritis were not troubled with recurrences it became evident that these must be due to some other cause. The discovery of the gonococcus in the prostatic secretions years after infection, in some cases even twenty or thirty years after, and in cases in which the possibility of re-infection could be excluded, has, however, explained the frequency of the recurrences as being due to the persistence of the infection in the system.

Tubercular iritis is frequently associated with cyclitis. In hospital cases, if the old letters have been preserved, we find sometimes records of repeated attacks of phlyctenular conjunctivitis. Instead of an ordinary iritis we sometimes find a number of small grey transparent tubercles in the iris. Still more rarely the tubercular infection assumes the appearance of a neoplasm, one or more granulomatous growths, or larger masses being found on the surface of the iris and may be unaccompanied with symptoms of iritis. Tubercular iritis is readily produced experimentally.

The other forms of iritis do not present any special features.

Sequelæ.—The more immediate sequelæ of iritis are the result of the inflammatory products found in the aqueous and in the iris. In the anterior chamber they not only considerably alter the composition of the aqueous, but, by clogging also the spaces of Fontana, may interfere with the normal passage of fluids from the eye, leading sometimes *to an increase of tension with a deep anterior chamber.* The exudates from the iris becoming organised may obscure vision if situated in the pupillary area (occlusion of the pupil), or may bind a portion (posterior synechia) or even the whole of the iris to the capsule of the lens (total posterior synechia), or the whole of the pupillary margin may be bound down, producing a total annular synechia (seclusio pupillæ). This is a very serious condition, shutting off the communication between the posterior and anterior chambers. The pressure of the aqueous which continues to be secreted by the ciliary body causes the iris to be pushed forwards (iris bombé) towards the cornea, and we have then *an increase of ocular tension with a shallow anterior chamber.* I would ask you, gentlemen, to bear in mind these two causes of increase of tension which you may meet

in the course of iritis because it is essential for the proper treatment of this complication.

The presence of synechiæ or remains of pigment on the lens capsule are very important aids in the diagnosis of iritis and especially of recurrent iritis, and it is necessary that you should not mistake for synechiæ the remains of pupillary membranes. Posterior synechiæ always pass from the edge of the pupil to the capsule of the lens, the remains of the pupillary membrane pass from the anterior surface of the iris to the capsule. If you use a mydriatic, if there are posterior synechiæ the iris does not dilate or dilates irregularly. The bands of a pupillary membrane are elastic and do not interfere with the proper dilatation of the iris—the patient may also help you by stating that the eye has been defective from early life.

Diagnosis.

The chief conditions from which you have to distinguish iritis are:

(*a*) Conjunctival inflammations.

(*b*) Small ulcers of the cornea.

(*c*) Acute glaucoma.

Conjunctivitis.—In conjunctivitis affecting both eyes there is generally no difficulty in diagnosis, but you may experience some difficulty in cases of severe unilateral conjunctivitis; the redness in conjunctivitis is general and extends from the the bulbar to the palpebral conjunctiva; in iritis the injection is at first ciliary, extending in severe cases to the conjunctiva, in these cases the changes in the iris should help you in the diagnosis. The character of the pain is of great assistance. The pain in conjunctivitis is of a burning itching character, with a sensation of discomfort, especially on moving the eye. In iritis there is severe ocular and supra-orbital pain, especially at night. In conjunctivitis the pupils are equal and react readily to light. In iritis the pupil of the affected eye is contracted and reacts sluggishly or not at all to light. In cases of doubt, if the pupil is small, we may put in a drop of homatropine and cocaine; if there is no iritis the pupil will dilate fully, but you should always warn the patient to come to see you again if the eye gets worse, for we may have had to deal with the very early hyperæmic stage of an iritis when the iris will still dilate under a mydriatic.

Ulcers of the cornea, in which there is an infiltration, can readily be seen with good illumination, but occasionally you find small clear ulcers with ciliary and conjunctival injection, and with a good deal of lacrymation and photophobia. The lacrymation and photophobia are much more marked than in cases of iritis, the pupils react readily to light, and the pain is more like that of a foreign body in the eye. A drop of a solution of fluorescein which stains the ulcer should make the diagnosis evident.

Acute glaucoma.—The chief points in diagnosis are: In glaucoma the pupil is dilated, inactive; in iritis it is contracted.

In glaucoma there is a shallow anterior chamber and there are no synechiæ, the cornea is hazy or streamy—due to œdema—the vision is considerably impaired early in the attack. The redness of the conjunctiva is intense, general, and of a dusky character; the pain is severe and may be accompanied by vomiting. The tension is considerably raised.

Tension, a dilated pupil, and a steamy cornea are the main points in the diagnosis of glaucoma. Glaucoma can be diagnosed by the finger. In acute glaucoma the tension is so much higher than that of the unaffected eye that there should be no difficulty in detecting the increase of tension, but I can quite understand that the uneducated finger may, even in these cases, be not sufficiently confident of its own diagnosis.

Treatment.

As in all diseases the indications are the removal of the cause and the treatment of the local condition, unfortunately we are not always able to deal with the former, and we are thus often obliged to confine our treatment to the local inflammation of the iris.

Severe cases of iritis should be treated indoors; when the acute symptoms have abated the patient may be allowed out with a bandage or shade. An attack generally takes four to eight weeks to subside.

Local.—Energetic treatment should he started as soon as the disease is recognised. We prescribe atropine in 1 per cent. solution or ointment at first, every four to six hours, until we obtain a satisfactory dilatation, and after three times a day until the inflamation subsides. By the dilatation of the iris not only do we compress the distended vessels in its substance, but we also rupture the

small exudates which pass from the iris to the capsule of the lens and thus prevent their being organised into dense synechiæ. Atropine also renders the pupil inactive and thus sets the iris at rest. It is seldom that we are justified in using atropine stronger than 1 per cent. Occasionally under our own supervision one or two drops of a 2 per cent. solution, or even a granule of solid atropine may be placed on the lower lid, but it is necessary to keep the puncta from carrying the tears into the nasal duct by everting the lids.

We are, unfortunately, sometimes prevented from continuing the atropine by the intolerance of some patients to the drug, producing sometimes toxic symptoms, at other times a follicular condition of the lids, or pain and a severe eczematous inflammation of the skin of the lids and face (atropine irritation). We are then obliged to substitute some other mydriatic, the one most commonly used being hyoscine in $\frac{1}{2}$ per cent. solution. If this also is not tolerated we are forced to treat the patient without the use of mydriatics.

Relief of pain is obtained by the frequent application of dry or moist compresses, or the frequent bathing with warm boracic lotion. In severe cases great relief of pain and amelioration of the iritis follows the application of a few leeches to the temple. Ophthalmic practice is one of the few branches of medicine in which this method of treatment is used, leeches are frequently used in the ophthalmic hospitals in London and on the Continent with most satisfactory results.

The importance of breaking down synechiæ should not be overlooked, because even if they are not extensive at first if there are subsequent recurrences they may leave the pupil more and more bound down until secondary tension supervenes.

I have already explained the various ways in which tension may occur in iritis. In the case where we have tension with a deep anterior chamber you will recollect that the elevation of tension was due to the inflammatory products interfering with the passage of the aqueous through the filtration angle, it is obvious that if you use atropine you will further add to these difficulties by the thickened iris further obstructing the passage of the intra-ocular fluids. On the other hand if there is tension with a shallow anterior chamber due to the iris being pushed forward against the cornea by the aqueous, which cannot pass through the pupil, then the obvious treatment is to push the atropine and by breaking the posterior synechiæ restore the passage between the posterior and anterior chambers, and if you succeed in this the tension will disappear. Now you will ask, what are we to do if in the former case we cannot use atropine? We first see what effect the stopping of the atropine has. If the tension is still high we may use eserine, but it is better to perform paracentesis, that is to let out the aqueous, which not only lowers temporarily the tension, but allows the exit of the altered aqueous.

If you are unable to break down posterior synechiæ by means of atropine, and the tension is high, in order to avoid secondary glaucoma and loss of the eye, operative procedures are necessary so as to restore the communication between the posterior and anterior chambers. I do not propose to deal with these operative measures.

Constitutional treatment.—Syphilitic iritis is practically the only form of iritis which is influenced by constitutional treatment. I need not dwell on the treatment of syphilis, but I may mention that iritis can be effectively treated by salvarsan or neosalvarsan and the injection repeated if after three weeks the Wassermann reaction is positive. In the interval between the injections the treatment with mercury and iodide is continued.

Gonorrhœal iritis.—There is no specific treatment for gonorrhœal iritis. Vaccines have been used but the result of this treatment has not hitherto proved satisfactory. Gonococcal or mixed vaccines may be used. Gonococcal vaccines, 50 to 100 millions, may be given every three to seven days; some use weaker vaccines, others starting with 200 millions weekly increase the dose every fortnight up to even 1000 millions. Sensitised vaccines by the method of Besredka are also used. Recently Nicolle and Blaizot have used a special vaccine, prepared at the Pasteur Institute at Tunis, consisting of 1 part gonococci grown on glucose-gelose, emulsified, well washed in a 7 per 1000 solution of fluoride of sodium, and 9 parts of synococcus similarly treated (synococcus is a coccus usually associated with the gonococcus), which they claim is atoxic and does not produce an intense reaction. They have used it intravenously in adults, the injections containing 250 millions repeated every second day and subsequently doubled in strength. They have also used it in ophthalmia neonatorum, the vaccine being injected into the muscles of the buttock. It remains to be seen whether a more extensive use of the vaccine proves to be as satisfactory as is claimed.

Tubercular iritis.—Tuberculin treatment has given, on the whole, very satisfactory results. In England tuberculin T.R. has been most frequently used, commencing with doses of $\frac{1}{10000}$ milligram or more. The injections are continued weekly for two or more months, thus the patient is kept constantly under the influence of comparatively small doses.

Iritis from a septic focus.—The removal of the cause has frequently been sufficient. Autogenous vaccines of 100 to 250 millions or a mixed staphylococcal vaccine may be used.

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We regret that owing to the war the number of pages must be diminished for a time.

VISUAL SYMPTOMS IN ASSOCIATION WITH LESIONS OF THE SPINAL CORD.*

By ARTHUR W. ORMOND, F.R.C.S.,
Ophthalmic Surgeon to Guy's Hospital.

It is necessary first of all to tabulate two or three questions which I propose to answer in this lecture:

Why should visual symptoms occur at all in diseases of the spinal cord?

What spinal cord diseases are accompanied by visual symptoms?

What is the relation of the visual changes to the various spinal cord diseases, that is to say, what is the pathological relationship?

The optic nerve is part of the afferent or sensory nerve system, and is comparable with the peripheral sense system.

If we trace the path by which a sensory impulse is conveyed from the foot to the brain, we find that it traverses three orders of neurones.

From the peripheral end plate the impulse is conveyed along the sensory fibres of the nerve to the ganglion cells on the posterior roots of the spinal nerves, through the posterior columns to the nucleus gracilis and cuneatus. This constitutes the neuron of the first order.

From these nuclei the impulse travels by the internal arcuate fibres and tract of the fillet to the anterior and posterior quadrigeminal bodies and optic thalamus, constituting the neuron of the second order. From these basal ganglia the stimulus passes to the cortex cerebri behind the rolandic area, thus forming the third order of neurones.

If now we trace the visual paths in the same way we find here also that three orders of neurones can be made out, but not perhaps with so great a certainty as in the case of the peripheral nerves. All visual stimuli pass through the retina from

* Lecture delivered at the Polyclinic.

before backwards and reach the visual purple, where they produce certain definite chemical changes. These chemical changes affect the most external layer of the retina, namely, the rods and cones, which are therefore the specialised end bulbs of the visual paths. The rods and cones are connected with the rod and cone bipolar cells forming the main mass of cells found in the inner nuclear layer, and constitute the first order of neurones. The second order consists of the ganglion cells of the retina with their dendrones connecting them with the first order, and their axones which form the nerve fibre layer, and the fibres of the optic nerve. This is the second order of neurones. The third order, therefore, in the visual system would be represented by those fibres which pass from the external geniculate body, anterior corpora quadrigemina and optic thalamus, through the optic radiations to the occipital lobes.

Now it is clear from this that the axons of the retinal ganglion cells are analogous to the intra-spinal axons, both being neurones of the second order and not analogous to spinal dorsal root fibres. These latter are represented by intra-retinal fibres, either those of the inner nuclear layer or those of the outer nuclear layer, but which, it is difficult to say. The ganglion cell fibres belong, therefore, to the central nervous system, and it is doubtful if such fibres ever regenerate. The ganglion cell layer is found to correspond to the cells in the nucleus gracilis and cuneatus; the optic fibres in the corpus quadrigeminum and optic thalamus correspond to the mesial fillet in the mesencephalon.

The answer, then, to the first question is this. The optic nerve corresponds to the white matter of the spinal cord; it consists of fibres which are the axons of the ganglion cells of the retina, just as the fibres forming the posterior columns are the axons of the cells forming the posterior root ganglion; consequently it is liable to the same pathological processes as is the cord itself. The optic nerve fibres from the ganglion cells to the basal ganglia, *i. e.* external geniculate and quadrigeminal bodies and thalamus, are associated with lesions involving the posterior columns and cerebellar tracts; the posterior columns represent the first order of neurones, and the cerebellar tracts the second order, and we must therefore modify to a certain extent our anatomical analogy and recognise this pathological fact, that the optic nerves and tracts are associated with the posterior columns and cerebellar tracts in their pathological lesions.

In order to make this point clearer I have arranged the path of sensory impulses through the posterior columns and cerebellum, and also the visual paths in parallel columns.

Sensory Path (through Posterior Columns).

Neuron of First Order.

(1) End-plate.
(2) Dendrones } of post. root ganglion cells.
(3) Nuclei }
(4) Axons = posterior columns.

Neuron of Second Order.

(1) Dendrones of cells in nucleus gracilis and cuneatus.
(2) Nuclei.
(3) Axons from these cells which pass through the internal arcuate fibres, tract of fillet to the anterior and posterior quadrageminal bodies and optic thalamus.

Neuron of Third Order.

Fibres formed by axons coming from cells in the quadrageminal bodies and thalamus to the cortex cerebri.

Sensory Paths via Cerebellum.

Neuron of First Order.

(1) End-plate.
(2) Dendrones } of post. root ganglion cells.
(3) Nuclei }
(4) Axons.

The axons terminating in arborisations around cells in Clarke's column.

Neuron of Second Order.

(1) Dendrones of cells in Clarke's column.
(2) Nuclei.
(3) Axons forming the direct cerebellar tracts to the cerebellum (middle lobe).

Neuron of Third Order.

(1) Dendrones of cerebellum cells.
(2) Nuclei.
(3) Axons to cortex cerebri.

VISUAL PATH.

Neuron of First Order.

(1) Sensory epithelium of retina (rods, cones, etc.).

(2) Dendrones } of bipolar cells.
(3) Nuclei }
(4) Axons.

Neuron of Second Order.

(1) Dendrones of ganglion cells of retina.

(2) Nuclei.

(3) Axons constituting optic nerve fibres which pass through the chiasma, optic tracts to the external geniculate body, anterior quadrigeminal body and optic thalamus.

Neuron of Third Order.

Fibres formed by axons coming from nuclei in quadrigeminal body and thalamus, through the optic radiations to the occipital cortex.

The second question may be answered by giving the list of diseases of the cord which have visual symptoms associated with them :

Tabes dorsalis, certain familial and hereditary ataxias, such as Friedrich's disease, cerebellar ataxy, disseminated sclerosis, myelitis, tumours and injuries.

The occasional record of optic atrophy or neuritis with other spinal cord diseases is to be looked upon as due either to coincidence or the presence of some secondary factor present in the same case.

The answer to the third question, that is, the pathological relationship, must be considered in the case of each disease separately.

TABES DORSALIS.

In 1904 a gentleman, æt. 45 years, came to consult me because he had lost his colour sense. He had been a man of considerable activity, a Mayor and a person of great local influence in his native town, but lately had had what he termed a nervous breakdown, and he was passing through London on his way to Switzerland in order to recuperate. His visual acuity was bad and was only slightly improved by spectacles. He could see to read large print and had about six diopters of myopia. His colour sense had completely gone. The pupils were unequal, but reacted to light and accommodation. The knee-jerks were absent, the fields of vision very contracted, and the optic discs atrophic. He was a non-smoker and tee-totaller, but on inquiry I found he had contracted syphilis when about 19 years old. A diagnosis of tabes dorsalis was made and was confirmed by Dr. Fred Batten. I saw him on two subsequent occasions and found Argyll-Robertson pupils present. He got steadily worse and died quite blind in 1911.

In tabes dorsalis we are dealing with a primary progressive *degeneration* of the first order of neurones, that is, with a degeneration of the prolongations within the spinal cord of the cells of the posterior root ganglia.

These prolongations consist of: (*a*) fibres which form the posterior columns (the first order of neurones); (*b*) fibres which surround the cells of Clarke's column, and from which fibres pass to form the direct cerebellar tract (the second order of neurones); and (*c*) those fibres which form the spinal reflex arc.

The degeneration is seen best in the posterior columns, and examination shows that the myelin sheaths of the nerve fibres are first diminished and then destroyed. The axis cylinders after being altered, swollen, and irregularly attenuated, become completely degenerated. The neuroglia is increased, and certain secondary changes take place in the blood-vessels. In other words the degeneration is of the same nature as is that produced by section of the posterior roots.

How do the changes in the optic nerve and tracts compare with these changes in the spinal cord? The pathological changes in the optic nerve in tabes have been described as follows (Parsons):

"The general consensus of opinion as to the pathological anatomy of the optic atrophy of tabes is that the primary seat of the disease is in the retina, commencing in the ganglion cells and nerve fibre layer. In the nerve, the atrophy is generally most marked near the globe, diminishing towards the cerebrum; changes in the chiasma and tracts are absent in the early, and are slight even in the later stages. The visual tracts above the so-called primary optic centres are not affected.

"Degeneration of the nerve fibres commences with breaking up of the myelin sheaths. Later the axis cylinders become varicose and degenerate.

"The retina shows marked degenerative changes in the ganglion cells and in the nerve-fibre layer without any signs of inflammation or proliferation."

You see from this comparison that the pathological changes found in the spinal cord and in the optic nerve are the same, and that both involve the axons of ganglion cells.

What are the visual clinical symptoms found in tabes? An atrophy, commencing as a central scotoma, a concentric contraction of the field, or a combination of these; once started, it is usual for the disease to progress until both optic nerves are atrophic, leaving the patient totally blind in from two to four years. I have had cases in which the earliest visual symptom was a relative central scotoma for red and green, simulating an ordinary tobacco amblyopia; others again in which neither the central or peripheral field had failed, but irregular islands of defective vision occurred, scattered about in the fields. Atrophy is the rule and has no exceptions. Where neuritis has been recorded in cases of tabes, the coincidence is due to the presence of a syphilitic or other cause. Atrophy occurs in about 10–15 per cent. of cases of tabes. It may be the first symptom, preceding any other by long periods of time, even years. Benedict stated (1887) that the tabetic motor symptoms vanished when atrophy of the nerve occurred. This is certainly not true, but we do find many cases in which the atrophy of the optic nerve is marked and the ataxic condition slight, and *vice versa*.

Mott finds post-mortem atrophy of the nerve fibres in many cases in which it was not diagnosed during life. His observation was made amongst patients in asylums, where there are inherent difficulties in examining patients with the ophthalmoscope. He puts the percentage of cases of tabes with optic atrophy at 50 per cent., but his patients were mostly observed when the disease was progressing to a fatal issue. It is probable therefore that the percentage of cases of tabes with optic atrophy is much larger than 10 to 15 per cent. which I have given, if we take cases in which the clinical history is complete; few cases seen among the out-patients of a general hospital are traced to the end. I need not recount the ophthalmoscopic picture of optic atrophy because it is given in every text-book, but let me remind you that pallor of the optic disc is not evidence of defective vision, since the palest disc I ever saw had no atrophy. The visual acuity and the size and mobility of the pupils are better guides and should be noted in every case.

We find visual symptoms in those familial and hereditary ataxias which have, as an essential lesion, either a primary *degeneration* of certain afferent tracts to the cerebellum, or of the cerebellum itself; frequently the dorsal columns of the spinal cord are also involved. The best known of this type is Friedreich's disease.

This is a familial disease starting between the fifth and fifteenth years of life. Optic atrophy is rare, but has been observed by too many writers to allow us to suppose that it is only a coincidence. Pathologically it is due to a primary atrophy of the proto-neurones pertaining to the muscle sense, the posterior columns and cerebellar tracts being involved.

Spino-cerebellar ataxia is more frequently associated with optic atrophy than Friedreich's ataxia. In this disease the cerebellar tracts are involved more extensively than the posterior columns.

Cerebellar ataxy due to primary degenerative disease of the cerebellum is so rare that little is known about it, but optic atrophy has been recorded as existing in one case.

Various visual symptoms have been described in association with sub-acute diffuse degeneration of the spinal cord, or Gower's ataxic paraplegia, optic atrophy, optic neuritis and retinal hæmorrhage. It is a multiple focal disease with secondary systemic degenerations, due to an involvement of the white matter of the cord by patches of degeneration; the medullary sheaths degenerate and the axis cylinders disappear. The posterior columns are usually the first to be affected, and later on the whole of the white matter may be involved. Little is known of the cause of the disease, but it is frequently associated with anæmia, diabetes, etc.

In all these diseases of the spinal cord, then, which are due to a degenerative change in the posterior columns or cerebellar tracts we find a primary optic atrophy without inflammatory signs.

Insular Sclerosis.

In insular or disseminated sclerosis we find both optic neuritis and optic atrophy. The neuritis precedes the atrophy as a rule, but a failure of vision may precede any ophthalmoscopic evidence of change. If we study the pathology of this disease we shall see how this is brought about. In

disseminated sclerosis islets of sclerosis occur scattered about in the white matter of the spinal cord, brain, and cranial nerves. Large patches of sclerosis are preceded by acute vascular dilatation and inflammatory œdema ; this œdema is transient and clears up with comparative rapidity, but during the early acute stage it is characterised by its tendency to spread far beyond the inflamed centre. When it subsides there is left a patch of softening with the myelin sheaths destroyed, the axons are affected but slightly, and these are subsequently found in the mesh-work of neuroglia forming islets of sclerosis. There is no secondary degenerative process, since the axons escape, at any rate for a long time. The optic nerve represents a portion of a system of neurones which corresponds in every respect to other systems, such as the posterior columns of the spinal cord Its affections in the various diseases of the spinal cord are entirely analogous to the affections involving the posterior columns and other sensory afferent tracts.

When the posterior columns degenerate as a system, that is, as a whole, the optic nerve degenerates entirely, undergoing complete atrophy as in tabes dorsalis, but in disseminated sclerosis no system is affected as such, because insular sclerosis is not a systemic disease, but a scattered one. Ocular symptoms may appear at any time in the course of this disease, and may precede all other symptoms. Generally there are no ophthalmoscopic signs, or only slight transient ones, but in some cases the ocular signs are well marked.

The clinical symptoms vary very much. A patient may notice a slight blurring of his vision, or a rapid and almost total failure, then a rapid recovery, to be followed after a long period by permanent, partial, or total failure. These symptoms may be associated with optic nerve hyperæmia, definite optic neuritis, optic atrophy, or even no ophthalmoscopic sign at all. In the optic nerve patches of sclerosis occur most frequently immediately behind the eye-ball in the neighbourhood of the central retinal vessels, with less frequency in the intra-cranial segment of the optic nerve and in the chiasma. The lesion is identical with that found in the spinal cord, viz., a rapid and extending œdema, demyelinisation of the nerve fibres without secondary degeneration, persistence of the axis cylinders, then after a time neuroglial sclerosis and destruction of the axis cylinders if the change is sufficiently severe.

It is clear, therefore, that the pathological findings explain the clinical symptoms. A patch of sclerosis occurring some little distance behind the eye-ball might temporarily obscure vision, without any ophthalmoscopic change being observable, or at the worst a transitory hyperæmia, and the axis cylinders remaining intact; the visual impulses could be transmitted. If, however, the pathological focus was close to the eye, then the optic disc would show definite œdema and hyperæmia similar to that described by Mr. Leslie Paton in the 'Trans. Ophth. Soc.', vol. xxxiv, p. 252. The recovery of vision is due to the absence of any secondary degenerative changes and to the escape of the axis cylinders. If, however, the pathological changes progress, the axis cylinders may subsequently be destroyed and permanent failure of vision result. In disseminated sclerosis the visual fields vary; constriction of the periphery may be present, or a central scotoma relative or absolute. Other changes which have been described are annular scotomata, quadrants or irregular sectors missing, and transitory amblyopia. Total loss of sight, that is, permanent, rarely ensues, or complete hemianopic areas either. Bullock concludes, as the result of some experimental work on this subject, that the disease is due to an infection by an ultra-microscopic organism, or to a poison which may be found in the central nervous system, and which is soluble in water. This virus is supposed to be the ultimate cause of the various changes found.

Myelitis and Optic Neuritis.

An acute myelitis is often preceded by some febrile disease, as typhoid, smallpox, measles, diphtheria, scarlet fever, or gonorrhœa, and is possibly due to the toxins of the diseases, or the local presence of the micro-organisms themselves. We do not, however, possess any complete knowledge of the ætiology of the trouble, although it is generally assumed that the lesions in the cord and optic nerve have a common origin which act directly upon the nerve substance.

If the grey matter alone is affected we term it poliomyelitis, and in that case no visual symptoms are to be expected, the optic nerve fibres corresponding, of course, to the white matter only.

The pathological changes found in the nerve and cord are similar, and are of an inflammatory nature. The inflammation is somewhat localised in vertical extent and involves more or less all the tracts, irrespective of their nature or origin; it may be acute or sub-acute, an acute myelitis being associated with an acute neuritis, a sub-acute myelitis with a sub-acute neuritis. All the signs of inflammation may be present: vascular dilatation, extravasation of red cells, and leucocytic emigration. But there is not the extensive œdema found in this disease that is so characteristic of insular sclerosis. If the inflammation is severe, the axis cylinders are entirely destroyed; in slighter cases resolution takes place.

Taylor and Collier assert that whenever the optic nerves are affected the cervical enlargement of the cord is involved, and the optic nerves do not suffer if the myelitis is confined to the dorsal or lumbar regions; in other words, the changes in the optic nerve have some relation to the contiguity of the inflammatory process in the cord.

A case of myelitis with neuritis has recently been under my care at Guy's Hospital. A young man attended the out-patient department, suffering from most of the symptoms of tobacco amblyopia. He was not a smoker, however, and seemed to be otherwise quite "fit." A week later he had difficulty in walking, and ten days afterwards developed weakness in both legs and arms, and lost control over his bladder and rectum. The visual symptoms became more marked, and both discs appeared to be slightly neuritic. For three months he was prostrate and was unable to move his legs; his arms were also affected. He recovered, however, and in twelve months' time from the date of the onset of his illness he was quite well, had regained power, and had good visual acuity.

The changes found in vision may be (*a*) nil; (*b*) a retro-bulbar neuritis; (*c*) optic neuritis with œdema; or (*d*) atrophy.

Vision may be affected by the lesion too far behind the eye-ball to permit definite ophthalmoscopic signs. The presence of a central scotoma points to an involvement of the periphery of the nerve. A neuritis with well-marked œdema shows that the inflammatory focus is close to the eye-ball, but the "neuritis" is usually not at all prominent, being generally only associated with a slight haziness of the disc margin, and an increase in the number and size of the vessels.

The atrophy is secondary, following the destruction of the axis cylinders by the severity of the inflammation, but here again the ophthalmoscopic picture may vary very little from the appearance found in a primary atrophy if the preceding neuritis has been far behind the globe.

Both eyes are generally involved, but not equally or at the same time.

A large number of these cases are fatal, the percentage having been estimated as high as 50 per cent.

Tumours and Injuries.

Visual symptoms have been described in association both with tumours of the cord and also with injuries.

Taylor and Collier state that optic neuritis of all degrees of severity may occur in connection with tumours, hæmorrhage, or compressions of the cord.

Clifford Allbutt first drew attention to the changes in the optic nerve in injuries of the cord, and stated that the higher the lesion the more frequent were there optic nerve changes. No cases are recorded in which the nerve is involved when the lesion is below the second dorsal segment.

It is difficult to explain the occurrence of the changes, that is to say, what are the pathological conditions which lead to it, but probably they are due to secondary changes of the nature of a myelitis which occur in the cord as a result of injury or compression.

In conclusion, then, we find (*a*) that visual symptoms are associated with certain lesions of the spinal cord; (*b*) that the optic nerve belongs to the second order of neurones, and consists of the axons of the ganglion cells formed within the retina; (*c*) that the optic nerve is part of the central nervous system, and reacts pathologically in the same way as the white matter of the cord, being subject to the same pathological processes; (*d*) that when visual lesions are associated with spinal cord disease, the posterior columns are usually affected, and less frequently the cerebellar tracts.

January 11th, 1915.

FATAL HÆMOPTYSIS IN A CHILD AGED FOUR YEARS.*

By J. D. ROLLESTON. M.D.,

AND

J. E. ROBERTSON-ROSS,

Assistant Medical Officers, Grove Fever Hospital, London.

A BOY, æt. 4 years, was admitted to the Grove Hospital on February 19th, 1914, certified to be suffering from whooping-cough. Measles, in December, 1912, was stated to be the only previous illness, but since November, 1913, he had appeared generally unwell and had been subject to attacks of abdominal pain and vomiting.

Condition on admission: The child was in a very ill-nourished and wasted state. The cough was paroxysmal, but at no time during his stay in hospital was he heard to whoop. Rhonchi and *râles* could be heard all over both lungs, but no areas of dulness could be marked out. The abdomen was distended and the superficial abdominal veins were dilated. The spleen could be palpated below the level of the costal margin, and the area of liver dulness was slightly increased downwards. No enlargement of the lymphatic glands was detected.

The temperature rose nightly to between 102° and 103° F., but fell to nearly normal in the morning. The pulse-rate was rarely below 120, and the respirations varied between 30 and 50.

On March 15th a specimen of sputum was with difficulty obtained from the mouth, but no tubercle bacilli were seen. Von Pirquet's reaction, performed on April 3rd, was also negative, a fact which is of interest in view of the subsequent post-mortem findings. (A positive reaction, however, was found in the patient's sister, æt. 2 years, who was admitted on the same day with the certificate of whooping-cough, and died on May 21st of tuberculous meningitis, a terminal episode of what was probably a generalised infection. An autopsy could not be obtained.) A differential blood count on April 4th showed: Polymorphonuclears, 54 per cent.; small mononuclears, 40 per cent.; large mononuclears, 6 per cent.

Very little change occurred in the child's general condition, but on April 13th impairment of resonance was noted below the left ninth rib behind and extending round into the left axilla; over this area the breathing was bronchial in character.

During the evening of April 24th a very little blood stained sputum was expectorated, and at 8.30 p.m. on April 25th, after a comfortable day, during which the child had seemed rather brighter than usual, a sudden copious hæmoptysis occurred, death ensuing almost immediately.

Necropsy (April 26th): On pressing on the trachea and œsophagus a quantity of dark blood was expelled from the mouth and nostrils. The tracheo-bronchial glands were much enlarged, obviously tuberculous and some of them caseating. The left pleura was thickened and firmly adherent to the chest-wall and diaphragm. The upper lobe of the left lung was studded with tuberculous deposits. The lower lobe was solid, small pieces of it sinking in water, and presented a cavity about the size of a walnut, completely filled with recent blood-clot. The whole of the right lung was a mass of tuberculous nodules. The liver was enlarged, fatty, and studded with tubercles. The spleen was considerably enlarged and contained tuberculous deposits of a fair size. Tubercles were also seen in the kidneys, basal meninges, and grey matter of the cerebral cortex. The heart was empty and normal in size and appearance. The mesenteric glands were enlarged and the stomach contained a large quantity of altered blood, but showed no sign of any lesion in the mucous membrane.

This case is of interest in the first place as an example of general tuberculosis with the primary focus in the tracheo-bronchial glands producing a paroxysmal cough simulating whooping-cough; and in the second place owing to the rarity of hæmoptysis in so young a child. Although, as in seven out of twenty-three cases collected by Meusnier which came to autopsy, the actual bleeding vessel was not definitely identified, it is presumable that the hæmoptysis was due to rupture of a branch of the pulmonary artery into the cavity found in the left lower lobe. Death was ascribed to asphyxiation rather than to the amount of blood lost.

A somewhat similar case occurred at the same

* Read at the Royal Society of Medicine.

hospital nearly three years previously. A boy, æt. 4 years, was admitted with the certificate of whooping-cough on June 8th, 1911. His cough was sometimes paroxysmal, but no whoop occurred during his six months' stay in hospital. The signs and symptoms were those of chronic pulmonary tuberculosis, and the paroxysmal cough suggested affection of the tracheo-bronchial glands. Von Pirquet's reaction, performed on July 19th, was positive. Two attacks of profuse hæmoptysis occurred on December 20th, and death took place the same day. No autopsy could be obtained.

Meusnier, in 1892, collected thirty-three cases of hæmoptysis in children aged from 8 days to 14 years, but only sixteen of these were in children under 5 years. Of these sixteen all but three were tuberculous and all but one—a case of influenzal bronchitis—were fatal. The other two non-tuberculous cases occurred in newborn infants, in one of whom the lungs seemed to have been digested by the inspiration of gastric juice during vomiting, while in the other the hæmoptysis was due to pulmonary thrombosis of syphilitic origin. Magruder, in 1908, described a case in a negro boy, æt. 3 years, very similar to our own, and referred to four other cases aged from 7 months to 3½ years in which fatal hæmoptysis was due to pulmonary tuberculosis. Other cases of fatal hæmoptysis in children due to pulmonary tuberculosis have since been reported by Brown in a girl æt. 3 years, and by Thomas in an infant æt. 3 months.

It would thus appear that hæmoptysis occurring in young children is, in the majority of cases, due to pulmonary tuberculosis. It is, as a rule, a sudden and fatal event. In only five of Meusnier's twenty-nine fatal cases had the terminal hæmoptysis been preceded by any spitting of blood. In this respect the slight premonitory hæmoptysis which occurred in our case was exceptional.

The hæmorrhage may be caused by the rupture of an aneurysm or of a branch of the pulmonary artery into a cavity within the lung, either in the apex or the base, or by the breaking-down of tracheo-bronchial glands surrounding a vessel and causing a rupture into a neighbouring bronchus. The fact that the mouth, pharynx and lower air passages are as a rule filled with blood, demonstrates the mode of death in these cases. The stomach is generally found to contain much blood, suggesting at first sight hæmatemesis, but no lesions can be detected in the mucous membrane. Other causes of fatal hæmoptysis in children besides tuberculosis may be gangrene of the lung, ulceration of the neck vessels in scarlet fever, or gangrenous angina, or hæmorrhage in ulcerative sore throat, an example of which was reported by one of us to this Section during the last session.

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January 11th, 1915.

MODERN ANÆSTHETICS. By J. FREDERICK W. SILK, M.D.Lond., Senior Anæsthetist and Lecturer on Anæsthetics, King's College Hospital. London: Edward Arnold. 1914. 3*s.* 6*d.* net.

This book opens with a history of the general and local anæsthetics and is followed by the general phenomena attending anæsthesia. The chapter on the choice of the particular anæsthetic in special cases such as nasal and cleft palate operations, and in the "status lymphaticus," and the duration of operation, is well written and constitutes a safe and efficient guide to the practitioner who has often to weigh and ultimately decide the particular anæsthetic, sometimes alone, at other times in consultation with the surgeon. Detailed information is given on the administration of the general and local anæsthetics, as well as the after-effects such as shock, sickness and the like, while the methods adopted in exceptional operations are extremely useful. The chapter on regional and local analgesia with a most complete index conclude a book which no general practitioner can afford to be without.

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We regret that owing to the war the number of pages must be diminished for a time.

A LECTURE

ON

ANTE-PARTUM HÆMORRHAGE.*

By THOMAS G. STEVENS, M.D., F.R.C.S.Eng., M.R.C.P.Lond.,

Obstetric Surgeon to St. Mary's Hospital, Paddington, and Queen Charlotte's Lying-in Hospital.

GENTLEMEN,—The subject of ante-partum hæmorrhage is of considerable interest to all of us, whether engaged in general practice or acting as specialists. Very few cases in the whole range of obstetrics give us greater anxiety or tax our resources more than the serious cases of ante-partum hæmorrhage, and particularly those of the type of concealed accidental hæmorrhage.

I do not propose to go over with you the whole subject of ante-partum hæmorrhage, but I want to mention a few points which may be of interest, and which are more or less novel, particularly in relation to the very serious types of cases which one meets with, only too frequently. Of course, a man who is in general practice will only meet with ante-partum hæmorrhage occasionally. He may go on working for a couple of years before he meets with a case of placenta prævia; but when these cases do occur they may tax his resources to the utmost.

Let me just clear the way by giving one or two definitions. We can divide cases of ante-partum hæmorrhage into accidental and unavoidable: accidental hæmorrhage meaning bleeding from the separation of a normally situated placenta; having nothing to do with such accidents as blows, falls or shocks; accidental only because the bleeding occurs from a normally situated placenta. On the other hand, we speak of unavoidable hæmorrhage because the bleeding occurs from the separation of a placenta which is situated in the lower uterine segment. When the placenta is situated in the

* Delivered at the Polyclinic.

lower uterine segment bleeding must occur when labour begins, because the placenta must separate as the *os uteri* opens out. In the case of accidental hæmorrhage the blood naturally finds its way out of the uterus and passes between the membranes and the uterine wall. This is known as external accidental hæmorrhage. But under some conditions the blood is retained between the membranes and the uterine wall, or between the placenta and the uterine wall, in which case we speak of it as "concealed hæmorrhage." In the case of placenta prævia the hæmorrhage is always external, never concealed.

There are one or two points which occur to me with regard to the causation of these conditions which we must not overlook. Why is it that certain women are predisposed to separation of the normally situated placenta? What kind of women are they? As a rule, these are women who have had several children, they are generally middle-aged, and of bad general health. If you go into the histories of a number of cases of accidental hæmorrhage, you will nearly always find albuminuria associated with the condition, an albuminuria which is not permanent, but which generally clears up after delivery, and fairly quickly after delivery. In other words, it appears that in these cases, whatever it is which causes the albuminuria, the same cause is also responsible for the hæmorrhage. The probability is that the placenta separates because there is a small initial hæmorrhage between the placenta and the uterine wall. The placenta does not separate, in these cases, from any external cause, although the patient, in many instances, will be able to find some circumstance which she supposes was the cause of her trouble. She may tell you that she slipped off a chair, or that she fell down-stairs, or that she has had a great shock. But if you go into these events you generally find that these different circumstances do not bear any direct relationship to the separation of the placenta. As it appears to me, the initial factor is a small hæmorrhage between the placenta and the uterine wall; and then, perhaps, slight uterine contractions are set up, because the small hæmorrhage acts as a foreign body. With the contractions the blood is squeezed out, separating more and more of the placenta, and gradually making its way to the outside, between the membranes and uterine wall. There is another feature which has been brought prominently to our notice of late years, and that is the frequency with which hæmorrhage into the uterine muscle occurs in cases of accidental hæmorrhage. There have been cases described in which, after death, or at operations, the uterine muscle has been found absolutely infiltrated with blood. Further than this, cases have been described in which the uterine muscle and serous membrane have actually given way under the influence of this hæmorrhage; bleeding has then occurred into the peritoneum, or into the connective tissue of the broad ligaments, showing there must be considerable pressure of the effused blood.

No one at present has been able to give a satisfactory explanation of this intra-muscular hæmorrhage, but taking into account the almost constant occurrence of albuminuria with it, I cannot but feel that the whole is a manifestation of some profound form of toxæmia, allied, no doubt, to the toxæmia which causes eclampsia, and to the toxæmia which causes albuminuria, and possibly allied to that which causes pernicious vomiting. It is only another manifestation of the toxæmias of pregnancy. But it is an interesting one in this particular case, because it occurs in women who have had many children, whereas the other toxæmias of pregnancy occur, as a rule, in first pregnancies.

Turning to the local condition of the uterus in accidental hæmorrhage, it is nearly always the case that the patients are subjects of the condition which has been known as chronic metritis. We all of us, perhaps, have some kind of perception of what we mean by that term; it is usually a grouping of clinical symptoms associated with a local lesion of the uterus, rather than an actual histological picture of the lesion. For instance, we think of a married woman who has had several children, who suffers from leucorrhœa, menorrhagia, and backache, who has no displacement, but who has a hard and what I speak of as a "poker-like" uterus, because it is very stiff and straight. This woman has a condition which may be described as chronic metritis, and which has also been called fibrosis of the uterus. The case sometimes comes under the heading of endometritis if the lining of the uterus is thickened; but we always mean the same thing, namely, a patient who suffers from that collection of symptoms which I have mentioned. These women, if they become pregnant, are predisposed to accidental

hæmorrhage, to placenta prævia, and to early abortion. But it is difficult to see what relationship there can be between such an uterus and that of any condition of toxæmia which must be something outside the actual local histological changes in the uterus. Recently, it has been shown that the uterus in chronic metritis does not contain, as a rule, an excess of fibrous tissue, but an excess of elastic tissue, particularly in the walls of the blood-vessels, and in and around the muscular bundles themselves. The amount of elastic tissue in a virgin uterus is comparatively small, and is limited to the inner coat of the arteries. But a uterus the subject of chronic metritis contains a very large amount of elastic tissue, a fact which can be easily shown by means of special staining reactions. I only mention this as a point of interest, not as an attempt to explain to you why hæmorrhage occurs in such cases.

The causation of placenta prævia, the development of the placenta in the lower uterine segment, is still a mystery. The old explanations—one of which was that the fertilised ovum, arriving in the uterus from the Fallopian tube, dropped to the lower part of the uterine cavity instead of being retained in the upper part—must be given up as untenable. It is more reasonable to suppose that the actual site of the development of the placenta in such cases is partly on the decidua capsularis, causing the placental site to descend lower and lower in the uterine cavity as the embryo enlarges.

Thus we arrive gradually at the varieties of placenta prævia, which are central, partial, and marginal—central when the placenta completely covers the internal os, partial when it partially covers over the internal os, and marginal when it only just reaches the edge. There is none of the mystery attaching to placenta prævia, however, that there is attaching to the manifestations of accidental hæmorrhage. When once the placenta has developed in the lower uterine segment, nothing can prevent hæmorrhage taking place when labour begins. As soon as the os uteri opens up the placenta cannot stretch to keep pace with the size of the internal os, consequently it must separate; when it separates bleeding must occur from the uterine wall. The placenta itself does not bleed, and the child loses no blood, because, of course, the chorionic villi, which belong to the fœtus, are not torn when the placenta separates.

The next point I want to bring to your notice is one of considerable clinical interest, and that is, the relationship of the uterine contractions to the severity of the hæmorrhage. When we consider cases of accidental hæmorrhage, one of the things which strikes us as of great importance is the extreme danger in which the patient is placed when there are no uterine contractions. Picture to yourself a placenta separating, or partly separating from the uterine wall. Blood is being poured out, in great or in small quantities, and finds its way between the membranes and the uterine wall to the outside, or remains inside. What is there to stop that bleeding? There are two factors: uterine contractions and coagulation of the blood. If the blood coagulation is in any way at fault, that factor will fail. If there are no uterine contractions, the torn blood-vessels cannot be compressed, consequently there is nothing to prevent the patient bleeding to death, unless she can, in some way, be relieved. On the other hand, if the uterus is contracting moderately or powerfully, there must always be a certain amount of compression of the blood-vessels, and therefore the bleeding will stop spontaneously. That is the reason why in so many cases of ante-partum hæmorrhage the bleedings are only slight, and occur only for a short time, and hence require no special treatment. The uterine contractions, and, above all, the uterine retraction, which accompanies the contractions, close the blood-vessels and stop the hæmorrhage. The worst cases of accidental hæmorrhage are those in which there are no uterine contractions at all. Concealed accidental hæmorrhage is the worst, most dangerous, and most fatal accident which can happen to a pregnant women. It is, for instance, far more fatal than eclampsia, or than post-partum hæmorrhage, or than any other toxæmia. And the reason for this fatality is that it can occur only in women whose uteri fail to contract. It is as if the uterus were paralysed, and the blood poured out between the membranes and the uterine wall gradually stretches the uterus until it becomes hard and distended, the stretching going on until it is possible for the patient to bleed to death into her own uterus. Since we have known a little more about this condition we have seen that hæmorrhage into the muscle occurs all over, and in such cases it is not difficult to see why the muscle is paralysed. Muscle infiltrated with blood is probably cut off

temporarily from the nerve endings by pressure, or by stretching; and consequently stimuli which normally would pass to the uterus and make it contract fail now to reach their object. Of course I am now picturing to you the very worst type of case. The moderate cases do not show hæmorrhage to any great degree into the uterine muscle, and they do not present any very great difficulties in the matter of treatment.

But the treatment of concealed hæmorrhage has been a puzzle to obstetricians in all ages, and even now the question is not settled. We see precisely the same danger in cases of placenta prævia in which the uterus will not contract. We know that it must have contracted to some extent in order to have caused bleeding at all, because the placenta will not separate unless there are some uterine contractions, but the contractions need to be very slight, and may be only those painless contractions which occur all through pregnancy, and which may just suffice to open up the internal os, so that it will, perhaps, just admit the finger-tip. Such uterine contractions will not be appreciable to the patient, but they will be sufficient to cause separation of the placenta, and possibly severe hæmorrhage. We see, further, that the harder the uterus contracts, the more the placenta will separate in a case of placenta prævia. It does not follow, in a case of accidental hæmorrhage, when the uterus contracts hard that the placenta separates any more. It may not. But with all uterine contractions in placenta prævia the placenta must separate until the os is fully dilated and will allow some part of the embryo to come down.

Therefore in placenta prævia, although we must have had uterine contractions, they are, in themselves, a source of danger; whereas in accidental hæmorrhage they are a source of only good to the patient.

Another point I should like to make is as to the difficulties of diagnosis. The commonest kind of case of ante-partum hæmorrhage that comes before us is that of a multiparous patient, who complains that she has been having slight hæmorrhage, somewhere about the seventh or eight month of pregnancy. Possibly you will have a history that she has had one rather big hæmorrhage. She may not have taken any notice of these hæmorrhages; or possibly she may have gone to the doctor at once about them. When you see a patient like that, you cannot help saying to yourself "This is a case of hæmorrhage from partial separation of the placenta; is it accidental, or is it unavoidable? Is it a normally situated placenta, or is it placenta prævia? Does it really matter which it is?" Yes, it does matter. It matters in this way: the majority of cases of accidental hæmorrhage are, fortunately, slight, and can be treated by simple means. But *all* cases of placenta prævia are dangerous; and no woman with placenta prævia is safe until she has been delivered. She may not be safe even then, because she may get post-partum hæmorrhage. But, at the same time, there is no spontaneous cure for the hæmorrhage of placenta prævia, whereas there is a spontaneous cure for hæmorrhage which comes into the category of accidental. Therefore we have to make up our minds, when we see a women bleeding who is pregnant, whether her case is one of placenta prævia or whether she has a separation of a normally situated placenta. This recognition is not always easy. If, however, the cervix uteri is sufficiently dilated to permit the introduction of a finger, you may be able to feel the placenta, and the case is settled. Such a woman must be delivered, and no temporising measures are permissible. But if you cannot feel the placenta, if you cannot reach it with the finger passed through the canal of the cervix, the case may still be one of placenta prævia, because the lower edge of the placenta may just reach that portion of the uterus which Herman called "the dangerous zone," that is to say, the lower segment of the uterus, which has to dilate so as to allow the child to pass through. But we have this comfort when the placenta only just reaches the danger zone, that the case is never one of any very great severity, and the hæmorrhage will, nearly always, cease as the os dilates. The more the placenta covers the internal os, the more danger there is to the patient. Therefore in all cases of ante-partum hæmorrhage it is important to try to pass the finger through the cervical canal, to see if the placenta can be felt. If the canal is not open enough for the finger to pass through, then the case is difficult. Are you going to dilate the cervix so as to allow your finger to pass through? There is one point which may be of some value. When the placenta occupies the whole of the lower uterine segment, as it would do in any serious case, we find that the presenting part will not sink down into the pelvic cavity; the placenta prevents that, so you

will always find the presenting part above the brim; and that would be evidence that something rather than a child was filling up the lower uterine segment; it would be presumptive evidence in favour of placenta prævia, and not of a normally situated placenta. That will therefore decide, if you find such a thing, that you must make your internal examination so as to find out where the placenta is. But if the presenting part—the head for choice—has entered the brim, and has sunk deeply into the pelvis, I think you may take it for granted that the placenta cannot fill up any considerable portion of the lower uterine segment; and in such a case you might be justified in waiting, if the hæmorrhage were not severe. You might leave such a case alone if there were only slight hæmorrhage. But you cannot leave any case alone in which the hæmorrhage has once been severe.

I should like now to touch on the methods of treatment of moderate cases of accidental hæmorrhage, and of moderate cases of placenta prævia. I need not go over the old-fashioned methods of treatment; I will only mention those which are in use now and which are practicable.

Let us take external accidental hæmorrhage first. I shall take it for granted that the finger has been passed through the internal os, and that no placenta can be felt; and yet you have made up your mind, owing to the severity of the hæmorrhage, that the patient must be delivered. There are two definite methods of treatment, which are somewhat opposed to one another. One of them is an older method, namely, rupturing the membranes artificially, putting on a tight binder, and, if the patient has had many children, giving some drug which will excite uterine contractions. Ergot is a drug which used to be recommended for this, but now we recommend pituitary extract for the purpose. This is, perhaps, the oldest method of treatment for accidental hæmorrhage which can be mentioned, and it is still applicable to mild cases, if any treatment is required for them at all. I think it has a very small applicability, because the cases which are of mild degree will nearly always cease to bleed spontaneously if they are left alone, or if they are kept very quiet; whereas cases which are severe will not be successfully treated by this method. The other method of treatment is the Dublin plugging method, about which a great controversy has raged, and which is not settled even now. It resolves itself into this: that under some circumstances tight plugging of the vagina, along with tight bandaging of the uterus, beginning the bandaging from above and forcing the uterus down upon the vaginal plug, with a perineal pad and bandage tightly applied, will, under some circumstances, excite uterine contractions, will stop hæmorrhage from a normally situated placenta, and will lead to delivery in a reasonable number of hours, without danger to the patient. The originator of this method claimed that the plug and binder actually compressed the uterine arteries—a desideratum which I could never bring myself to believe—was thus accomplished. One knows how difficult it is to compress an artery with one's finger when there is a hard substance, such as a bone, to compress it against. But there is nothing hard in the pelvis against which a uterine artery can be pressed; and therefore it is unlikely that any pushing of the uterus down to a plug could possibly compress the uterine arteries. But it sets up irritation and starts uterine contractions, and if uterine contractions can be started in accidental hæmorrhage, the bleeding ceases almost at once. These two methods—the plug and binder and the rupture of membranes—are applicable only, I take it, when the cervix is not dilated and when the membranes are unruptured. With the plug and binder the membranes must be kept intact, because we want to get the pressure inside the uterus raised as much as possible. If the cervix is dilated, and is, we will say, of the size of half-a-crown, it follows that there are efficient uterine contractions going on, and that the case could be treated in an entirely different way, namely, by bi-polar version, bringing down a leg, and then allowing the uterus to expel the child, after, perhaps, giving a small dose of pituitary extract. Cases in which the cervix dilates by itself with ante-partum hæmorrhage are mild cases, because uterine contractions are present. The patient may have lost a considerable amount of blood up to that point, but we have no control over that unless we have seen the patient all the time. A patient may lose an amount of blood which is fatal in ten minutes, from accidental or from unavoidable hæmorrhage, and over those cases we have no control. But a patient who is in labour, with good uterine contractions and with the cervix dilating, will not lose any significant amount more blood, because she has efficient uterine contractions.

Turning to the very severe cases of accidental hæmorrhage, amongst which we must include those of concealed accidental hæmorrhage. I imagine a patient who is suffering not only from blood loss, but also from shock. She is blanched, suffers from air-hunger, and has a very quick, small pulse, showing she has lost much blood. If you examine the abdomen in such a patient you find the uterus hard and resistant, tender to the touch because it is stretched, you can feel no fœtal parts, and you can detect no fœtal heart sounds, because the child is dead. Perhaps no blood has escaped externally, and you can infer that the patient has bled at all only from her general condition. What is to be done in the case of a patient like that, who is in such a desperate condition? If you rupture the membranes, and let off the liquor amnii, the pressure in the uterus will be reduced, and will allow further bleeding to take place. The bleeding may have temporarily ceased—you cannot tell that it has, but it may have, because the pressure in the uterus may have become equal to the blood-pressure, in which case, theoretically, no more bleeding should occur. But directly the membranes are ruptured the intra-uterine pressure is diminished, and further bleeding will take place. If the cervix is undilated, you cannot dilate it under a certain number of minutes. You cannot carry out any form of accouchement forcé without running great risks of injury to the lower uterine segment. Dilating bags are useless because the paralysed uterus is unable to contract. Finally, during the time consumed by any of these methods, the patient may go on bleeding, and may die before delivery can be effected. If you use the plug and binder method, you will probably be no better off, because the pressure in the uterus is already as high as you can get it, and you can get it to a higher point only if the uterine muscle begins to contract. But I have shown you that the uterine muscle is paralysed and stretches before the increasing pressure of the blood-flow. Therefore the plug and binder method is not likely to do good in either stopping hæmorrhage or in initiating uterine contractions. We are therefore forced to conclude that some surgical operation must be performed, and the question arises, What? The patient is in a desperate condition from blood loss and from shock. If by your surgical operation she loses more blood, the probability is that she will die. Of course, it has been suggested—and carried out—that Cæsarean section is the proper treatment for concealed accidental hæmorrhage in a severe case. It has been done successfully; it has been done unsuccessfully. But one can see at once a reason for the non-success of Cæsarean section in some cases, because no man can guarantee to perform Cæsarean section without a certain amount of blood being lost. The uterus is already full of blood, and directly you open it of course the blood gushes out, and further bleeding occurs from the placental site. Remember you are dealing with a uterus which will not contract, and one which is infiltrated with blood and paralysed. Therefore my view is that Cæsarean section is not the operation of election for a bad case of concealed hæmorrhage. The proper operation in such a case is hysterectomy, removal of the uterus without any attempt to interfere with its contents. By doing hysterectomy in such a case you can guarantee that the patient will not lose another couple of ounces of blood, and you cannot guarantee that by any other operation. You do not open the uterus at all, but simply remove it entirely. I have had the opportunity of performing this for concealed hæmorrhage, and I have done it for central placenta prævia, and I was exceedingly pleased with the result in both instances. I shall mention those more fully presently. You can see that to carry out any form of surgical procedure in a patient like this, who is already in the most dangerous condition that a woman can be in, requires some precautions. These precautions are: first of all, saline infusion whilst performing the operation, and secondly, the use of the smallest quantity of a general anæsthetic combined with local anæsthesia of the abdominal wall.

Saline infusion is best carried out by means of Lane's infusion bag; a large rubber bag to contain five pints of salt solution, which has a single tube, ending in a two-way tube, with a hollow needle in in each portion. A needle is inserted under each breast, and the bag is hung up five feet above the patient. By this means one can get 1½ pints under each breast in thirty or forty minutes.

Anæsthesia is best induced by the use of a combination of drugs. First, morphia and atropine hypodermically, a quarter of a grain of the former and a hundredth of a grain of the latter. Then the middle line of the abdomen should be infiltrated in three layers with ·25 per cent. solution of novocain,

and in many people the operation can now be carried out without any general anæsthetic at all. But as the patient is probably frightened, as well as in a severe condition of shock from blood loss, it is wise to use a small amount of general anæsthetic. I recommend the smallest amount of ether, given by the open method, which will just keep the patient asleep. This will suffice if the abdominal muscles have been properly infiltrated with ¼ per cent. novocain solution. The operation of hysterectomy, performed under those anæsthetics, is very safe, and can be done in about twenty-five minutes by a quick operator. And at the end of the operation the patient should be no worse than at the beginning. You may say that this is no operation for the man who is not accustomed to doing abdominal surgery. That is quite true; it is not. But I am sorry to have to say I cannot offer you any alternative method of treatment which can be expected to save a life in a severe case of concealed hæmorrhage. Naturally, much depends on the amount of blood which has been poured out into the uterus. If it is only a small amount, then by stretching the cervix with bags, with the fingers, or with a Bossi dilator, you may be able to perform version, and extract the child by force. By this means you might be able to save an occasional patient, but you will not be justified in *expecting* to save your patient by this means if she has lost a considerable amount of blood, because she will lose even more after delivery, and she will die of post-partum hæmorrhage. And there is another risk which we know she must run, and which must have killed many patients in the past without our knowing it. I refer to rupture of the fibres of the uterus and of the peritoneum, and hæmorrhage into the peritoneal cavity. We are only finding these things out now that we perform abdominal operations for severe cases of hæmorrhage. We did not know them before because we very seldom had the opportunity of performing a post-mortem examination on a woman who died in child-birth. Very few cases have been examined, except in lying-in hospitals, in cases in which death has occurred from concealed or accidental hæmorrhage.

So, from every point of view, if a patient can be got to a hospital, or if the services of a trained abdominal surgeon can be obtained, that must be the method of treatment for concealed hæmorrhage. There is no reason for hesitation in removing the uterus, because the uterus of a patient who has once had concealed hæmorrhage is only a source of danger to her.

Now let us turn to placenta prævia and its treatment. Here I shall have to end up by telling you the same thing: that abdominal operations have sometimes to be performed in severe cases. I do not want you to think I consider abdominal operations are the only methods of treatment of accidental or unavoidable hæmorrhage; the operation is to be performed only in certain specially selected cases.

The common methods of treatment of unavoid able hæmorrhage are only two, but they are in the power of anybody to carry out.

The first method can be carried out without instruments, and with no apparatus except the operator's hands, that is to say, bi-polar version—bringing down a leg, and then carrying out slow extraction of the child. Naturally, this operation can be successfully performed only when the cervix is slightly dilated and will admit two fingers, and when the placenta is partly situated over the lower uterine segment. It is not easy to perform bi-polar version when we are dealing with a central placenta prævia. If you can reach the edge of the placenta it is easy enough to do version. The half breech is brought into the lower uterine segment, presses against the bleeding uterine vessels, and so the hæmorrhage is stopped. Therefore it is a satisfactory and successful method of treatment for what I may call mild cases of unavoidable hæmorrhage.

The second method of treatment of placenta prævia is by Champetier de Ribe's bag. It is a large bag, having a circumference of at least thirteen inches at the widest part. It is a rigid bag, having a silk basis covered over with rubber. It is easy to put it into the lower uterine segment when the os will admit one big finger easily or two small fingers.

On the whole, the use of this bag is better than bi-polar version and extraction, because it gives the child a better chance. As soon as the bag is put in and filled, it stops the hæmorrhage, because it compresses the lower uterine segment, and bleeding cannot take place. You can attach a weight to it so as to produce some degree of pull, and increase the pressure in that way. But it has one great danger, namely, that the natural expulsion of the bag by uterine contractions may be followed

immediately by a very severe or even fatal hæmorrhage. Therefore, if you are going to use the bag, be present when the os uteri is almost completely dilated, and take the bag out; do not let it be expelled. As soon as it comes out, slip your hand into the uterus and do version, or put on the forceps and bring the head down. In those ways you will prevent hæmorrhage, and will probably secure a good result. We have still to face the chance of post-partum hæmorrhage in placenta prævia, because the placenta, being largely in the lower segment, is attached to a part in which contraction and retraction are inefficient. Now that we have pituitary extract, the bug-bear of post-partum hæmorrhage is almost done away with. The effect of pituitary extract lasts only a certain number of minutes, forty or sixty. Therefore, if you give a full dose, you must be ready to deliver in thirty-five to forty minutes. If you can get delivery completed, then the third stage of labour will usually be concluded without risk. But if 1½ to 2 hours are going to be allowed to elapse before the delivery is effected, the state of your patient may be as dangerous as if you had not given any pituitary extract.

What is to be done for the severe cases of central placenta prævia in which the patient has lost much blood? Let me relate a recent case.

A patient was admitted to Queen Charlotte's Hospital in July, 1914, having had a severe hæmorrhage. She was treated by a Champetier de Ribe's bag. The bleeding ceased, but in spite of that, the patient remained with a pulse rate of 140. She was pale and looked bloodless, and had obvious air-hunger. By using saline and employing warm bottles and various restoratives, we managed to improve her condition, so that her pulse dropped to 120. A decision as to treatment had to be made. There was nothing in the vagina, and the bag had been removed. She was having a certain amount of uterine contraction, in fact, the uterus had been in a state of tonic contraction. Directly we made a vaginal examination she began to bleed again. The cervix was nearly fully dilated, and was almost the size of an ordinary tea-cup. But she could not have been delivered in less time than five or ten minutes, and we could not guarantee that she would not lose another pint of blood when the placenta came away. The patient had had nine children. The present fœtus was already dead, so I did not have to consider it. I therefore determined to perform a panhysterectomy without interfering with the contents of the uterus. The operation only took twenty-five minutes, and the patient made an absolutely uninterrupted recovery. I have since had the opportunity of doing the operation again, in a case of concealed hæmorrhage, a case in which the patient had what might have been a fatal hæmorrhage, but in which, fortunately, bleeding was stopped and restoratives applied. Then came the same question. How can she be delivered without bringing about a further blood loss so severe as to kill her? She had had several children, and this child was already dead, so again I performed panhysterectomy without opening the uterus. The result in this, as in the first case, was that the patient got perfectly well without manifesting any bad symptoms.

I do not think either of those patients could have been delivered *per vaginam* without further blood loss, and if either of them had lost any considerable quantity of blood they would have died.

I do not want you to feel that hysterectomy is the only treatment of placenta prævia, but I think that in certain cases in which there has been very severe blood loss which has stopped, or which has not stopped—and more so if it has not stopped—hysterectomy is the only treatment which will stop it effectually without further loss.

January 18th, 1915.

PRACTICAL MEDICAL ELECTRICITY: A HAND-BOOK FOR HOUSE-SURGEONS AND PRACTITIONERS. By ALFRED C. NORMAN, M.D. London: The Scientific Press, Ltd. 5*s*. net.

This book shows how far electricity has invaded the sphere of therapeutics. After a lucid explanation of the elements of that science, batteries, etc., the author passes on to its uses in every-day practice. The portable outfit for retinoscopy and the modification of the ordinary ophthalmoscope in favour of the Morton Marple variety, designed by Dr. Sutherland, deserves wide recognition both for its simplicity in general practice, but specially for those who devote their duties to estimate refraction in school children. Ionisation for the removal of corns might usefully find a place in a useful book.

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We regret that owing to the war the number of pages must be diminished for a time.

URINARY ANTISEPTICS.*

By J. W. THOMSON WALKER, F.R.C.S.,
Assistant Surgeon to St. Peter's Hospital.

A urinary antiseptic may be defined as a drug administered by mouth with the object of producing an antiseptic action in the urine. Urinary antiseptics act under great disadvantages as compared with antiseptics used in surgical work. The urinary antiseptic passes through two selective organs, the intestine and the kidneys, and for a time circulates in a powerful oxidising agent—the blood—before finally arriving at the seat of bacterial growth in a solution of uncertain concentration.

Antiseptics act by their affinity for the protein of the bacteria. In the diseased urinary tract the bacteria are suspended in a protein-rich medium, and the bactericidal substances combine more readily with the protein in the urine than with that of the bacteria. The urinary antiseptic is thus handicapped in its action by a protective covering around the bacteria.

The ideal antiseptic would be one which is rapidly absorbed from the intestinal tract and is non-irritating at the point of absorption, which is so stable as to remain unchanged in the blood, which is excreted entirely, or in large part, by the kidneys, and is non-irritating to these organs, and, finally, which will exert a powerful antiseptic action on the bacteria commonly found in the urine without, at the same time, causing irritation of the urinary passages.

By far the most important urinary antiseptic in use at the present time is hexamethylene tetramine. This is so generally known under the proprietary name of urotropin that I shall refer to it under this shorter name.

This drug more nearly approaches the ideal urinary antiseptic than any other, for it contains

* Lecture delivered at the Polyclinic.

in an innocuous form, which passes unchanged through the intestine and kidneys, a powerful antiseptic which is liberated in the urine. I propose to devote the time at my disposal to discussion of this drug.

I. Urotropin as an Antiseptic, and some Conditions which affect its Composition.

Urotropin is a condensation produce of ammonia and formaldehyde, and was first introduced as a urinary antiseptic by Nicolaier. An aqueous solution (ten per cent.) to which a little ammonia has been added to prevent the splitting off of formaldehyde, has no antiseptic action on cultures of the typhoid bacillus, *Bacillus coli*, or on staphylococci.

Urotropin dissolved in pure tap-water forms a slightly alkaline solution, and shows no tendency to decompose. Tablets which have been exposed to the air for some months show deliquescence, and on dissolving in water the reaction is acid, and free formaldehyde is present in abundance.

Urotropin is frequently prescribed with acid sodium phosphate, and I shall later refer to the significance of this combination. If the mixture be tested, free formaldehyde in large amount will be found. The same decomposition of urotropin is found in all combinations with acids, whether in the form of dry, compressed tablets, or in solution. The importance of this must, I think, be obvious. Urotropin owes its value as a urinary antiseptic to the fact that the formaldehyde which it contains is embodied in a form which is readily absorbed by the intestine, which causes no change in the blood, and has no deleterious effect on the kidney during excretion. It is only at the seat of infection, namely, the urinary tract, that formaldehyde should be liberated and exercise its irritant and antiseptic effects. You will see, therefore, how important it is that a preparation of urotropin should be fresh, and that hexamethylene tetramine should not be combined with an acid, or prescribed in a mixture containing an acid. Should it be desired to increase the acidity of the urine during the administration of urotropin, sodium acid phosphate should be given separately or the urotropin should be prescribed with ammonium benzoate.

Urotropin is rapidly absorbed from the intestine, and the greater part of the drug is excreted by the kidneys. It has also been found in the bile and pancreatic secretion, in the sputum, and in the secretion of the nasal sinuses and middle ear. It can be detected in the cerebro-spinal fluid obtained by lumbar puncture, and in the synovial fluid. In regard to the antiseptic value of urotropin in these alkaline media, I have very considerable doubt.

In the acid urine, in which most of the urotropin is excreted, the drug is split and formaldehyde is given off.

There is no longer any doubt that the antiseptic power of urotropin in the urine is entirely due to the splitting off of formaldehyde. The difficulties that hampered the earlier investigators in this line were due to the fact that the tests at their disposal did not discriminate between urotropin and free formaldehyde. This difficulty no longer exists, for the Rimini-Burman test, to which I shall refer later, demonstrates the presence of free formaldehyde and distinguishes between this and formaldehyde in combination as urotropin.

By the use of this test it will be found that under favourable circumstances free formaldehyde is present in the urine of patients taking urotropin.

According to a rough estimate by Burman, the administration of 10 gr. of urotropin thrice daily will give a solution of about 1 in 5000 or stronger of formaldehyde in the urine. This may, conditions being favourable, suffice to destroy an infection of the urinary tract. On the other hand, the infection may persist in spite of the continuous antiseptic action. I have seen a bacilluria develop in a patient suffering from stricture during the time that he was taking 30 gr. of urotropin daily and formaldehyde was being freely liberated in the urine.

I shall now refer briefly to the excretion of urotropin in a normal individual. After a dose of 10 or 15 gr. of urotropin the drug or formaldehyde can be detected in the urine in about an hour, but it is frequently delayed longer than this, two or even three hours being not unusual in my experience.

The excretion is at its maximum during a period of from four to eight hours after administration. After about twelve hours the excretion is still going on in small quantities.

I have detected formaldehyde in the urine seventeen and even twenty-two and a half hours after a single dose of 15 gr. of urotropin.

It would thus be possible, by taking two doses

of 15 gr. in twenty-four hours and passing water as seldom as possible, to keep the urine in the bladder continuously mixed with a strong solution of formaldehyde. In diseases of the bladder in which frequent micturition is a symptom, the period during which the bladder is under the influence of formaldehyde is reduced to about six or eight hours, the time during which the drug is being excreted in quantity by the kidneys. In such cases small doses frequently repeated (7 gr. every four hours) will be more effective in giving a continuous antiseptic action.

I may say a few words in regard to this question of dosage. Five or 10 gr. taken three times daily is the commonly prescribed dose. An increase in the dose to 15 or 20 gr. three times daily will be followed by an increased output of formaldehyde in the urine.

There is a limit to the increase in dosage, which in some individuals is very quickly reached. Urotropin itself is non-toxic, but the liberation of a high percentage of formaldehyde in the urine gives rise to irritation of the urinary tract. Irritability of the bladder, frequent micturition, and eventually strangury may occur. Hæmaturia has been recorded in a fairly large number of cases.

These symptoms have been known to follow small doses (2½ gr.) in certain individuals, but this idiosyncrasy is fortunately uncommon. Most patients can take 15 or even 20 gr. three times a day without undue irritation. On the whole, I am inclined to prescribe larger doses than is customary, and this is especially the case where I am dealing with an infection of long standing. It is better, I think, to produce a powerful antiseptic action, and do it for a short time, than to be content with a feeble action spread over a long time. The symptoms of an overdose of urotropin are, after all, not of a very alarming or of a permanent character, and can be controlled by ceasing the administration of the drug and giving an alkaline mixture. Twenty-five or 30 gr. repeated every four or six hours for twenty-four or forty-eight hours may suffice to cut short an infection which will drag on for weeks or months with smaller doses. It is a wise precaution before giving these larger doses to ascertain by smaller doses if the patient has any idiosyncrasy in regard to urotropin.

II. Certain Conditions which Influence the Action of Urotropin on the Urine.

(1) *The Effect of Variation in the Reaction of the Urine.*

(*a*) *Drugs which affect the reaction of the urine.*—It is necessary as a preliminary to the consideration of the effect of alkalies and acids upon the action of urinary antiseptics to discuss shortly the drugs which affect the reaction of the urine and the influence upon bacterial growth of variations in the reaction of the urine.

The normal acidity of the urine is due to the presence of acid sodium phosphate, and the acidity varies in different individuals and at different periods and times of the day under influences which are well recognised and need not detain us here.

There is seldom any difficulty in reducing the acidity of the urine and giving it a moderately alkaline reaction. The drugs which are commonly used for this purpose are potassium citrate and acetate and sodium bicarbonate. The latter drug, in my experience, is the most powerful.

It is a much more difficult matter to turn an alkaline urine acid.

There are two types of alkaline urine met with in practice. One is a faintly alkaline urine which deposits phosphates sometimes in large amount, but which, apart from the change in reaction, is normal in other respects; the second is a powerfully alkaline urine with ammoniacal decomposition in which there is an abundant growth of bacteria together with other abnormal constituents, such as mucus, pus, and blood.

In practice you will find that there are only two drugs which will render a strongly alkaline urine acid. These are acid sodium phosphate and ammonium benzoate. The former may be given in doses of 30 gr. thrice daily, and, if necessary, increased to 2 or 4 drm. thrice daily. In the larger doses it may cause diarrhœa. Ammonium benzoate is given in doses of 15 or 20 gr. thrice daily.

(*b*) *The effect of alkaline urine on bacterial growth.*—I shall now turn to the effect of alkalies upon urinary infection.

The treatment of urinary infection by the administration of alkalies has been specially used in the pyelitis of childhood. It has also been extended

to the pyelitis of pregnancy, and is occasionally advocated in adult pyelonephritis. The pyelitis of childhood is an infection of the renal pelvis with a pure culture of the *Bacillus coli communis*, and the alkaline treatment of these cases has been ably advocated by Dr. John Thomson, and is now the settled practice with the great majority of physicians.

Potassium citrate and potassium acetate, and sodium bicarbonate, are the drugs usually given, in doses of from 24 to 48 grains in twenty-four hours. Under this treatment the urine becomes alkaline either after a few doses or in two or three days. When this has been accomplished the symptoms subside—the temperature falls to normal, the drowsiness and mental oppression vanish, the pain ceases, and the frequent micturition and scalding disappear.

If the alkaline treatment is discontinued there is a tendency for the temperature to rise again, and the symptoms reappear. Resumption of the alkaline treatment is again followed by disappearance of the symptoms. Eventually the alkalies may be discontinued altogether without recurrence of the symptoms.

The remarkable improvement that is observed in these cases has led to the view that the development of the *Bacillus coli* is inhibited, or the growth destroyed by the action of alkalies. This view apparently receives support in the observation that, when the urine is decomposing and ammoniacal, the *Bacillus coli* is either absent or, if present, it occupies an apparently subordinate position. Bacteriologists, on the other hand, deny that any disinfectant, or even antiseptic action, is exercised by alkalies in strengths such as can be developed in the urine.

There appears to be here a direct conflict between clinical and bacteriological work.

If, however, the cases submitted to the alkaline treatment be examined bacteriologically when the symptoms have disappeared, it will usually be found that the *Bacillus coli* is still present in the urine.

The action of alkalies in pyelitis appears to me to be a neutralisation of the acid toxæmia produced by the *Bacillus coli*. The cures that are claimed clinically are not cures in the bacteriological sense, for the infection remains; only the symptoms which were due to the acids, or acid endotoxins, have disappeared.

I would suggest, therefore, that the proper treatment in all cases of *acute* urinary infection with the *Bacillus coli* is as follows:

There should first be a course of alkalies, and the urine kept alkaline, until some days after the symptoms have completely disappeared. The alkalies should then be omitted, and urinary antiseptics given in increasing doses. If the urine remains alkaline after the alkalies have been omitted, sodium acid phosphate, or ammonium benzoate should be given in increasing doses to render it acid and cause the breaking up of urotropin.

The antiseptic course should not be combined too soon lest there be a recurrence of symptoms.

(*c*) *The influence of alkalies upon the action of urinary antiseptics.*—It has long been recognised that the action of urotropin is more powerful in an acid than in an alkaline urine, that the combination of urotropin with sodium acid phosphate assisted its action, while the combination with alkalies retarded it.

With the recognition that urotropin acted only by the production of formaldehyde, the question of efficacy has turned upon the splitting of urotropin in an alkaline and in an acid urine.

A good deal of discussion has waged around this point. One of the chief difficulties has been the lack of an accurate test, which will distinguish formaldehyde from urotropin.

In Rimini's test modified by Burnam, free formaldehyde and urotropin can be differentiated.

Burnam's modification of Rimini's test:—The following three solutions are used: (*a*) phenylhydrazine hydrochloride, 0·5 per cent.; (*b*) sodium nitroprusside, 5 per cent; (*c*) sodium hydrate, saturated solution.

Three drops of each of the first two solutions are added to the urine, and a few drops of the hydrate solution poured along the side of the test-tube.

If formaldehyde is present, a deep greenish-black colour passes down through the urine and clouds the fluid. This rapidly changes to green, and fades through bright green to orange and pale yellow.

The urine and the sodium hydrate should be warmed to slightly above body temperature. This test will detect formaldehyde in urine in a dilution of 1 in 150,000, but does not show the reaction

with urotropin. According to Burnam, there is an intense dark blue colour, changing to green in solutions of 1 in 20,000 or stronger, and in solutions of less strength the first colour is an intense green. If these tests for formaldehyde are negative, a fresh sample of urine should be boiled after the addition of a few drops of sulphuric acid. The test is then applied, and if the formaldehyde reaction is now present the urine has contained urotropin.

Turning now to the question as to whether formaldehyde is split off from urotropin in an alkaline or neutral urine.

(*a*) There is, in the first place, the question as to whether urotropin is effective when prescribed with an alkali. Take first a normal individual with a sterile slightly acid urine, in whom formaldehyde is liberated in the urine with ordinary doses of urotropin. After the administration of 15 gr. of urotropin, formaldehyde appeared in the urine in two hours. Twenty grains of sodium bicarbonate were then administered. Half an hour later the urine had become alkaline and formaldehyde was no longer present, although urotropin was being excreted. Forty grains of sodium acid phosphate were given, and a further dose of 20 gr. two hours later. Three and a half hours later the urine was again acid and formaldehyde was present in quantity. In this case, on a single dose of urotropin the urine was made positive to formaldehyde, negative to formaldehyde, and positive to formaldehyde again by merely changing the reaction of the urine.

It will be seen, therefore, that the administration of an alkali, together with urotropin, will prevent the liberation of formaldehyde. This was illustrated in practice in a case under my care where the patient had undergone prostatectomy and cystitis was present.

He was taking 15 gr. of urotropin and the same amount of ammonium benzoate, and the urine was acid, and formaldehyde being freely produced. The urine suddenly became alkaline; the formaldehyde disappeared. On inquiring for a possible cause I found that the doctor in attendance had given a cough mixture containing sodium bicarbonate. When this was omitted the urine again became acid, and formaldehyde was liberated.

(*b*) Let me now refer to the administration of urotropin alone where the urine is already alkaline.

I have alluded to two clinical types of alkaline urine, one in which the urine is habitually slightly alkaline and cloudy with undissolved phosphates, and the second when there is decomposition of urea by the action of bacteria.

I should first like to point out that a normal individual who is taking urotropin regularly may produce formaldehyde at one part of the day and fail to do so at another. This is due to the varying acidity of the urine. The morning urine is the most acid, and in those individuals always shows formaldehyde, but the urine taken after meals, during the alkaline tide, may show urotropin but no formaldehyde. On increasing the dose or by adding an acidifying drug such as ammonium benzoate, formaldehyde is continuously present in the urine.

Phosphaturia in a mild form is extremely common, and it is not confined to brain workers and individuals of a nervous temperament. In such cases formaldehyde is not liberated, and sodium acid phosphate or ammonium benzoate must be combined with urotropin before any formaldehyde is produced.

In cases of alkaline urine due to bacterial decomposition, so long as the urine remains alkaline, no formaldehyde appears in the urine, and, therefore, no antiseptic action is obtained. The whole question of urinary antisepsis by urotropin in these cases turns upon our ability to render the urine acid. If we are unable to do so an antiseptic action cannot be obtained with the formaldehyde series of urinary antiseptics.

The degree of acidity necessary for the splitting of urotropin appears to vary somewhat. With litmus paper a faint tinge of red practically always means a negative formaldehyde, and a haze of phosphates has a similar significance. A well-marked red reaction with litmus will usually be definitely positive to the formaldehyde test.

When the total acidity is measured more accurately by means of a deci-normal soda solution, the point at which the formaldehyde reaction becomes positive varies somewhat. In one patient it is positive at $22\frac{N}{10}$ soda, while in another it remains negative until $32\frac{N}{10}$ soda is reached.

(To be concluded.)

January 25th, 1915.

FOREIGN BODIES IN THE EAR, NOSE, AND THROAT.*

By EDWARD D. DAVIS, F.R.C.S.Eng.,
Assistant Surgeon, Nose, Throat, and Ear Department, Charing Cross Hospital, etc.

THE presence of foreign bodies in the ear, nose, and throat frequently occurs, and the many practical points in connection with their detection and extraction, followed by the certain cure of the patient, makes the subject well worth consideration.

In the ear, *cotton wool plugs* which have been lost are the most common objects.

Patients with otorrhœa, for their own comfort, plug the ears with wool, and in some cases this is concealed by being pushed far into the meatus and is forgotten, or only a piece is removed, and the application of a second plug drives the wool further in.

The danger of the retention of such an object is obvious, but, if it is fortunately detected, removal by *ear forceps* and with a good light is easy.

Paper, beads, pebbles, or pieces of lead pencil are discovered accidentally, or the patient owns to having pushed such an object into the ear.

Occasionally such a foreign body, when it is near the drum, will produce a persistent and unaccountable *cough* or irritation of the meatus, but more often there are no symptoms unless the object is large enough to occlude the meatus and produce deafness.

In the extraction of such a foreign body the *anatomy* of the external auditory meatus is important. The floor of the meatus is formed by a mound or hillock whose summit corresponds to the centre portion of the meatus where the cartilaginous and bony meatus unite, and where it is narrowest. A foreign body which slips beyond this hillock may be concealed behind it and be more difficult to extract against the hill. On the other hand, when the object is being extracted with a hook, the hook is passed above it so as to keep it on the floor of the meatus, which is more easily seen than the roof, and the descending slope from the hillock to the orifice of the meatus facilitates extraction. Extraction, by placing the hook below the foreign body, is impeded by driving the foreign body out of sight into the roof of the meatus and against the overhanging edge of the orifice in this position.

Non-absorbent foreign bodies, such as beads or pebbles, are most easily syringed out, and the best syringe is a *Higginson* syringe with a curved cannula. The stream of boracic lotion is directed against the roof or posterior wall of the meatus to avoid the impaction of the lotion against the drum, which is painful and detrimental.

Objects such as peas or paper pellets, which swell up with lotion, should be extracted by a small ear *hook* or crocodile forceps under vision, and on no account should *blind* attempts at removal be made.

Foreign bodies in the nose are usually found in the middle meatus, and their retention leads to the characteristic blood-stained discharge from one nostril.

Objects shown as examples:

(1) A button impacted in the posterior nares for twenty-five years.

(2) A rhinolith.

Such objects are sometimes covered with calcium salts and form what is known as a rhinolith.

A loose foreign body in the nose may, during sleep or deep inspiration, fall directly into the larynx or trachea, and is more probable to do so than an object in the mouth, which is generally swallowed.

Extraction is carried out by cocaining the nose and by the use of a curved probe, hook or suitable forceps, under *vision*.

The position of a foreign body in the *pharynx* depends on the character of the object.

Pins, needles, fish-bones and sharp objects stick into the lower lobe of the tonsil, the base of the tongue or the pyriform fossa. Larger objects are impacted behind the cricoid cartilage or at what is known as the cricoid construction, a strong sphincter at the orifice of œsophagus, about six inches from the teeth. This is by far the commonest site for the impaction of foreign bodies.

If the article passes the cricoid, it is arrested in the gullet at the bifurcation of the trachea, ten inches from the teeth, where the left bronchus crosses the œsophagus and the aorta bulges in its outer wall, and if a foreign body is allowed to remain in this position ulceration may occur with fatal results. Finally, an object may be impacted

* Lecture delivered at the Polyclinic.

at the cardiac end of the œsophagus sixteen inches from the teeth.

In some cases, owing to a deep inspiration while a foreign body is in the mouth, the object may be inhaled and pass into the larynx, trachea, or bronchi, more particularly the right bronchus, which is larger, more in the direct line of the trachea and is not guarded as in the case of the left bronchus by the carina or tracheal septum.

In children foreign bodies appear to be tolerated in the larynx, trachea, or bronchi without causing suspicion. It frequently happens that a patient comes to the casualty with the history of having inhaled or swallowed something. There are no symptoms, and a superficial examination is negative; the patient is told to eat some bread or porridge and sent home. A foreign body in the larynx may produce laryngeal obstruction, and the condition resembles diphtheria or the various inflammations or paralyses of the larynx. The symptoms—hoarseness, aphonia, stridor, dyspnœa, etc.—are common to many such affections. If an object is impacted in a bronchus, an obscure chest condition arises in which the physical signs are limited to one lung—usually the right. A provisional diagnosis of empyema may be made, or even bronchitis and broncho-pneumonia, although these two latter generally have bilateral physical signs. In adults there may be an offensive purulent expectoration, with loss of weight, fever, etc., symptoms which resemble pulmonary tuberculosis. Bronchiectasis resulting from the inhalation of a foreign body has been known to simulate phthisis. Where unexplained symptoms and signs occur which may be due to an unsuspected foreign body, especially if they are unilateral and in a child, a careful history should be obtained and due importance attached to an account of the inhalation of something. Skiagrams should be taken and the bronchoscope employed.

The method of examination when a patient is alleged to have swallowed or inhaled a foreign body should be as follows:

Firstly, due importance must be attached to the history, to the character of the object and the sensations of the patient, who can sometimes localise the object.

Secondly, the mouth, fauces, pharynx, and larynx should be examined by the ordinary methods of tongue depression and laryngoscopy.

In the case of pins and fish-bones, particular attention is directed to the tonsils, base of the tongue and pyriform fossa. If a large object has been swallowed, a bulge may be detected in the neck in the line of the gullet, or if the trachea or bronchus is suspected, an examination of the chest may reveal physical signs.

Tympanitic resonance with absence of breath sounds on one side, particularly the right, are characteristic.

Foreign bodies have been detected in children by the fact that the signs were *unilateral* and asymmetrical.

Thirdly, an X-ray photograph is taken, but it must be remembered that even metal objects have escaped detection by skiagraphy. If there is any delay, the stools are watched for the passage of the object.

Finally, if the foreign body is still undiscovered, the pharynx, œsophagus, larynx, and trachea must be searched by suspension laryngoscopy, œsophagoscopy, or bronchoscopy. If the presence of a foreign body in the air or food passages is in any way possible, there should be no delay in searching for it.

Bougies or a probang more often than not fail to detect a foreign body. They also, unfortunately, push it further down, making it more inaccessible, and may even push it through the wall of the œsophagus. Bougies have also burst through the œsophagus into the pleura, particularly if ulceration has occurred. The coin-catcher has been known to tear the œsophagus, and in one case the instrument broke in the gullet, leaving the fragment behind, which caused the death of the patient. It is much simpler and safer to pass bougies or probes by the direct vision of the œsophagoscope.

Œsophagotomy for a foreign body should be extinct, as no difficulty has been experienced in extracting several tooth-plates, two of them having four teeth, by the same path as they entered, with the aid of the œsophagoscope. In the case of a firmly impacted tooth-plate there are scissor forceps for use with the œsophagoscope to cut up the plates to facilitate extraction. When pins are swallowed they are very commonly passed *per rectum*, but should always be found, for I have known them to have become impacted in the bronchus or œsophagus.

Owing to the ingenuity of Professor Killian, of

Berlin, and others, a foreign body can be extracted from almost any position in the air or food passages with negligible danger to the patient and consequent saving of life. The mortality of patients with foreign bodies in the air passages in the laryngoscopic period was as high as 30 per cent. (Killian). Since the introduction of the bronchoscope only 3 out of 182 died (Chevalier Jackson).

If the object is in the pharynx or larynx—*i. e.*, above the upper edge of the cricoid—suspension laryngoscopy is by far the easiest way of finding and removing it. One cannot be too enthusiastic in stating the merits of Killian's suspension laryngoscopy, more particularly for children, in whom a comprehensive and clear view of the pharynx and larynx is easily obtained and is decidedly better than that of the smaller tubes of the bronchoscope. To quote the words of a well-known laryngologist: "Suspension laryngoscopy makes laryngeal operations easy for the man in the street to perform." If the object is below the cricoid, the bronchoscope or œsophagoscope can be passed with the patient in the suspension position.

In the air passages particular attention should be paid to the smaller bronchioles of the right lung. For bronchoscopy and œsophagoscopy the largest tube possible should be used and its progress carefully watched, so that it is not allowed to slip past the foreign body, which in the case of the gullet may be hidden under cover of the lower edge of the cricoid cartilage, especially a flat object like a coin. In the œsophagus, at the bifurcation of the trachea, is another favourite spot of impaction, the gullet being slightly constricted by the aorta and left bronchus, a dangerous position for ulceration. The cardiac end of the œsophagus is also a probable site.

The technique of suspension laryngoscopy, bronchoscopy and œsophagoscopy is not difficult. The instruments are well-lighted specula which require gentle manipulation, and any surgeon could use them with success. Cases of impaction of foreign bodies are so common and the removal by the bronchoscope or œsophagoscope so efficient, that the method should not be confined to the specialist.

To illustrate the importance of accounting for the foreign body, I can quote a number of cases in which a mistaken diagnosis and delay in extraction has led to serious results:

Demonstration of: (1) *Suspension Laryngoscopy.*—(1) A safety pin extracted from the pharynx of an infant of 11 months; (2) A rabbit-bone removed from the larynx. Both objects shown were extracted by this method.

(2) *Œsophagoscopy.*—A number of coins and two tooth-plates removed by œsophagoscopy were shown.

(3) *Bronchoscopy.*

To summarise briefly: Foreign bodies in the ear, nose or throat should be extracted by vision and not by blind groping, such as the bougie, probang or coin-catcher.

A careful examination should be made whenever articles are alleged to have been swallowed or inhaled.

January 25th, 1915.

Necropsy in the Case of a Typhoid Bacillus Carrier.—Messerschmidt reports the case of an aged, mentally deficient woman, who in August, 1907, suffered from typhoid fever, following which she became a chronic carrier. In the year subsequent to the attack, not only the fæces but also the urine was infected. The urine then cleared up and the fæces continued to show intermittently positive results until the time of her death in 1912. Shortly after the original attack the patient had a purulent pleurisy, from which typhoid bacilli were cultivated. The bacilli persisted, however, in the pleuritic exudate for a few days only. At the necropsy typhoid bacilli were found not only in the bile but also the wall of the gall-bladder, in the liver, in the neighbourhood of the great blood-vessels, in the papillæ of one kidney, and also in one suprarenal capsule. As far as the latter organ is concerned, however, the condition is to be looked at as a secondary typhoid invasion of an old tubercular focus. As was to be expected, the bacilli were cultivated also from various portions of the intestinal tract. During life, treatment had been attempted with yoghurt, atoxylsaurem, mercury, and typhoid immune serum, but without result.—*Amer. Journ. of Med. Sci.*, cxlvii, p. 928.

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We regret that owing to the war the number of pages must be diminished for a time.

URINARY ANTISEPTICS.*

By J. W. THOMSON WALKER, F.R.C.S.,
Assistant Surgeon to St. Peter's Hospital.

(*Concluded from p.* 29.)

Let me now turn to the method of treatment in cases where the urine is strongly alkaline as a result of bacterial decomposition. It will be evident that the prescription of urotropin is useless until the reaction of the urine can be changed.

I have already stated that the only two drugs which act powerfully in turning an alkaline urine acid are sodium acid phosphate and ammonium benzoate. Sodium acid phosphate is given in doses of 20 gr. thrice daily, and the reaction of the urine carefully watched, the dose being increased every second day to 30, 40, 60, 90, 120, and, if necessary, 150 gr. thrice daily. The increase is limited by the effect of the drug on the bowel—when the dose reaches 360 gr. a little diarrhœa usually commences, and if the dose is increased will become troublesome. In the same way ammonium benzoate may be given in increasing doses of 10, 15, 20, and 30 gr. thrice daily.

The antiseptic drug which acts most powerfully in an alkaline urine is boric acid. The disinfectant action of this drug in the urine is very feeble, but it has a distinct antiseptic influence, that is to say, it prevents the development of bacteria. It has almost no effect on the reaction of the urine. A combination that is useful in alkaline bacterial urines is ammonium benzoate and boric acid, in doses of 10 or 15 grains of each.

This combination should, however, be used with the object of rendering the urine acid, and with this in view the dose of ammonium benzoate should be increased, and when the point of acidity necessary for the action of urotropin is reached, the latter drug should be added to or substituted for boric acid. The effect on an alkaline cystitis

* Lecture delivered at the Polyclinic.

of successfully turning the ammoniacal urine into an acid urine is remarkable, not only on the symptoms but also on the urine itself. A high-coloured, stinking urine, with a heavy slimy deposit of mucus and pus, loses its colour and smell and becomes clear, or almost clear, and the mucus deposit is reduced to a small layer or entirely disappears. The addition of urotropin at this stage frequently suffices finally to overcome the infection.

(2) *The Effect of Diluents and Dilute Urine on the Action of Urotropin.*

A not unimportant part of the routine treatment of urinary infections consists in the administration of diuretic drugs and waters. The method of inducing an excessive diuresis is either by the use of such drugs as citrate or acetate of potash, spirits of nitrous ether, theocin sodium acetate or other drugs, or by means of infusions such as parsley tea or buchu tea, or by large quantities of plain water, or by means of diuretic waters such as Contréxeville water, Vittel water, Wildungen water, and others.

Where the drugs render the urine alkaline their use in combination with the formaldehyde series of urinary antiseptics is on this account to be avoided, but where the action is largely that of diuresis, as in the teas I have named, and the diuretic waters this objection does not hold.

If an individual be placed upon urotropin so that formaldehyde is present in the urine and a draught of one of these waters be taken, the formaldehyde disappears from the urine and only returns when the diuresis has subsided.

We may, I think, conclude that an excessive diuresis such as is produced by these waters so lowers the acidity of the urine that splitting of urotropin does not take place. It is necessary, therefore, to choose between the two methods of treatment, namely: (*a*) powerful diuresis, and (*b*) antiseptic action by means of the formaldehyde series of urinary antiseptics. These two methods cannot be used concurrently.

In many cases of advanced disease of the kidneys a somewhat similar condition of the urine prevails—as, for instance, in the late stage of urinary obstruction from enlarged prostate or other cause, or extensive destruction from bilateral aseptic stone, or in bilateral hydronephrosis.

Urotropin is given in such cases in the simple faith that the excretion of urotropin and liberation of formaldehyde will go on as if the kidney were healthy. If we are to judge by experience with other drugs, such as potassium iodide, opium, methylene blue, or indigo carmine, we should at once conclude that this could not be the case.

Urotropin fortunately is non-toxic, and its accumulation in the blood, if the kidneys were too diseased to excrete it, would not have serious effects. At the same time it would be useless to give urotropin in advanced renal disease if it is not excreted.

From observations made on a number of cases of advanced renal disease, I have come to the view that there are two factors which reduce the efficacy of urotropin as a urinary antiseptic in such cases. First, there is diminished excretion of the drug in a few cases. A larger dose of urotropin is required before it is excreted in quantity in the urine. Secondly, when urotropin is well excreted, the condition of the urine in these cases is unfavourable for the liberation of formaldehyde. The urine is copious and neutral, or faintly acid, and it is difficult, and sometimes impossible, to increase its acidity.

At this point I may refer to the view held by Burnam, that the splitting of urotropin, and liberation of formaldehyde, is due to a vital action of the renal epithelium. The cases I have quoted might appear to support this contention. There are, however, many objections to this view. The dissociation is easily produced *in vitro* by the addition of acid and heat, and indeed it takes place, as I have shown, in any mixture containing urotropin which has an acid reaction. In the human body the splitting of urotropin can be influenced at will by turning the urine acid or alkaline, and it is extremely unlikely that a vital phenomenon could be so easily influenced in this manner. There is, I think, no doubt that the process of disintegration of urotropin in the urine is entirely a chemical one and depends upon the acid reaction.

(3) *Idiosyncrasies in Regard to the Formaldehyde Series.*

According to Burnam, not more than two patients out of every ten show decomposition of urotropin and liberation of formaldehyde in

the urine. This includes not only those suffering from urinary infection but also normal individuals. With small doses not more than 20 per cent. showed free formaldehyde; with large doses (20 or 30 grains repeated in four or six hours) 60 per cent. showed free formaldehyde. In a few cases, where formaldehyde was not present with doses of 30 grains, the dose was raised as high as 100 grains at a single dose without showing free formaldehyde. L'Esperance used the formaldehyde test in 250 patients, and found that only 50 per cent. of patients excreted formaldehyde. This was constant under all conditions of age, sex, disease, or health. The reaction of the urine was of no importance. Alkalies taken with, or in association with, urotropin had no effect on the production of formaldehyde, and increase in the dosage did not produce formaldehyde in negative urines. The figures given by L'Esperance are 130 (52 per cent.) positive to formaldehyde, and 120 (48 per cent.) negative. These results are attributed to idiosyncrasy of the patient.

I find it impossible to reconcile these statements with the results I have obtained. I have already shown in individual cases the extremely powerful effect of rendering the urine acid or alkaline—simple experiments that can easily be repeated by any observer. I shall now turn to larger numbers of cases. In 230 cases, all of which were given a dose of 10 grains three times daily, there were 79 (34·3 per cent.) negative to the formaldehyde test. Of these, urotropin was being excreted but not split in 64, and no urotropin was being excreted in 15. In the 64 formaldehyde negatives the urine was alkaline, or neutral in 52 and acid in 12. Of the 52 alkaline negative formaldehyde cases, 28 became positive when the urine was made acid. In 23 the urine did not turn acid under treatment, and it remained negative to formaldehyde. In one the urine turned acid, but the formaldehyde test remained negative. It will be seen, therefore, that the alkalinity of the urine was the dominating factor which prevented the splitting of urotropin. There were 12 cases of acid urine with negative formaldehyde. Of these, four were faintly acid, and became positive when the acidity was increased; four were faintly acid, but there was no opportunity of increasing the acidity; three were faintly acid, but were cases of renal insufficiency, and the acidity could not be increased; one was distinctly acid and negative, the urine being foul.

Passing now to the 15 cases where no urotropin was excreted. In eight cases a small dose was being given (5 or 10 grains), and when this was increased urotropin appeared in the urine. Three cases, with similar doses, could not be followed up. There was one case of advanced renal inefficiency, and one of profuse polyuria with very dilute urine. In two cases no cause for the absence of urotropin in the urine could be found.

Certainly in not more than five cases were we unable to explain on the lines I have stated the cause of the negative result of the formaldehyde test in the urine; three did not split urotropin in an acid urine, and two did not excrete urotropin at all. In regard to the three cases where the urotropin remained unchanged in an acid urine, I cannot give an explanation. The suggestion that such cases are due to an idiosyncrasy of the patient only, I think, partly explains the phenomenon. It is, I feel certain, due to some chemical peculiarity, and not to a vital action or the want of it on the part of the patient. The cases in which urotropin is not excreted at all in the urine can, I think, be explained in another and somewhat different fashion.

I have already shown how easily urotropin is decomposed and formaldehyde liberated, and how this splitting occurs in any combination in which the reaction is acid. It seems to me very likely that in these cases the explanation lies in the action of the acid gastric juice in splitting urotropin, and that the formaldehyde is not absorbed at all. It is well known that urotropin acts best when given with a large draught of water, and it is a matter of experience that the most powerful action is obtained by taking the drug between meals. In the first case the fluid probably dilutes the gastric juice and reduces its acidity, while in the second the time between meals is that in which a small amount of gastric juice is present in the stomach.

In order to obviate the splitting effect of the gastric juice, I have had urotropin put up in keratine capsules, which resist the gastric juice and guard the urotropin against its acid action during the time the drug is in the stomach. These capsules, manufactured for me by Messrs. Burroughs Wellcome & Co., should be used when urotropin appears scantily in the urine or does not appear at all.

III. Urinary Antiseptics as Prophylactic Agents.

There is nothing novel in the suggestion that I wish to make that urinary antiseptics should be used as preventive agents against urinary infection as well as in cases where urinary infection is already established. What I would like to see would be that this prophylactic action be considered seriously, not only when an operation upon the urinary organs is proposed, but also before all operations, and especially in operations which involve interference with the bowel or other pelvic organs.

Take, first, the most obvious cases where an instrument has to be passed into the bladder. The methods of sterilisation of urethral instruments are so simple and well recognised as to be within the reach of anyone, and are, I believe, widely practised. There are, however, conditions under which complete asepsis is peculiarly difficult, such as continuous retention after childbirth, when the operation must be delegated to a nurse, who may have to work under very difficult conditions and in unfavourable surroundings. And there are other cases where no slip in technique has happened, but urethral bacteria have passed with the instrument and find a ready soil in an obstructed bladder.

In all such cases, and in fact in all cases when an instrument is to be passed, an endeavour should be made to render the urine antiseptic by giving urotropin in doses of 15 gr. thrice daily and ascertaining by means of the tests already described if formaldehyde is appearing in the urine.

It will be necessary in cases where a catheter may possibly be required to anticipate its use by placing the patient on a sufficient dose of urotropin. Similarly in all operations upon the urinary tract formaldehyde should be present in the urine before the operation, and continuously afterwards until the patient is convalescent.

There is another class of cases which I think is worthy of closer attention than it has yet received. I refer to cases of post-operative urinary infection when the operation is performed in some part other than the urinary organs. It must have been the experience of many surgeons that the success of an operation upon the bowel, or an excision of the rectus or a Wertheim's operation for cancer of the uterus, or a pile operation is marred by a urinary infection which may cause constant discomfort for years, and which may eventually prove fatal. In some of these cases the catheter has been passed for post-operative retention, and the onus of carrying the infection is, rightly or wrongly, placed upon the catheter, but in other cases no instrument has been passed. In such cases a precautionary course of urinary antiseptics with the full development of the formaldehyde action in the urine could not fail to exercise a powerful effect in deciding the issue of the post-operation infection —whether, that is to say, it would develop or be only a passing bacilluria. I would advise, therefore, that the urinary tract be prepared by a course of antiseptics, just as the bowel is prepared by aperients before all operations when interference with the bowel or pelvic organs is probable, and that this be made a routine before parturition.

IV. The Limits of Urinary Antiseptics.

Let me finally say a few words in regard to the limits of urinary antiseptics. I have already referred to the limitations that idiosyncrasy in certain patients places upon the dosage of urinary antiseptics. I shall now refer to another type of limitation in the use of these drugs.

In infection of the urinary tract the surgeon early comes to recognise two types. In one type the urinary infection is the only abnormal condition present in the urinary organs, apart from some reaction of the mucous membranes. In the second type some other urinary disease is present, such as stone, chronic prostatitis, or urinary obstruction (hydronephrosis and pyohydronephrosis, enlarged prostate, stricture). In the first class of cases the use of urinary antiseptics is frequently sufficient in itself to cure the infection. One type of case is, however, particularly troublesome in this class—namely, cases of pure bacilluria. In this kind of case, however, a gradual increase of the dose of urotropin, together with the provision of an acid urine in which it will split may be followed, first of all, by the appearance of light deposit of pus in the urine on standing, and then in clearing of the urine on standing. Finally, the great growth of bacteria begins to diminish and eventually disappears.

There are other cases where it is impossible to obtain a sufficiently high acidity of the urine to

produce splitting of urotropin. In such case, and in those where a systematic attempt is being made to render the urine acid by means of acidifying drugs, boracic acid is the best antiseptic drug that we possess. In all such cases local treatment by antiseptic washes must be used carefully and systematically. The extent of the infection and its principal focus must, first of all, be ascertained by means of cystoscopy and catheterisation of the ureters. Where the bladder alone is infected, this should be washed, and where pyelitis is present, lavage of the renal pelvis should be carried out.

Turning now to the second class of cases, where some definite pathological condition is present in addition to the urinary infection. In a patient suffering from enlarged prostate, with an undrained pool of infected residual urine, in a case of infected renal or vesical stone, chronic prostatitis, stricture, or pyonephrosis, there can be no hope of overcoming the infection by urinary antiseptics alone. It is necessary to remove the residual urine by catheter or operation, to remove the stone, to relieve the obstruction which causes the hydronephrosis, to cut or dilate the stricture, and to treat the prostatitis by rectal massage or other local measures. When this has been done urinary antiseptics will complete the cure by overcoming the infection.

February 1st, 1915.

Bone in the Sputum.—Kretschmer reports a case of this very rare phenomenon. A man, æt. 31 years, brought some pieces of spongy bone, which he said he had expectorated. He was found to have a pleuritic effusion, probably an empyema. In the sputum were elastic fibres. A slight kyphosis was found, corresponding to the last two dorsal vertebræ. The patient continued to expectorate bone while under observation. The first diagnosis was abscess of the lung with secondary erosion of the vertebræ, but this opinion was changed to a tuberculous abscess of the vertebræ with rupture of the same into the pulmonary tissue.—*Med. Record*, vol. lxxxvi, p. 1024.

ACUTE PARAPLEGIA.*

By ANTHONY FEILING, M.D., M.R.C.P.,
Assistant Physician to the Metropolitan Hospital and the Hospital for the Paralysed and Epileptic, Maida Vale.

MR. PRESIDENT AND GENTLEMEN,—The subject of my address is one which must sooner or later be brought before the notice of every practitioner of medicine. Acute paraplegia—by which I mean a paralysis of the lower limbs of rapid onset—is an event which must always be terrifying to the patient and his friends, and sometimes equally so to the doctor. For one of the first questions to be asked will certainly be "How soon shall I recover the use of my legs?" or "Shall I always be paralysed?" Now this question must be answered, and we cannot do so nor even attempt any adequate prognosis at all until we have made an accurate diagnosis.

So that the diagnosis of such a case, so far from being a matter of mere academic interest, assumes a very real and practical importance. We must therefore be familiar with the various pathological processes which can cause this condition, and, further, have some criteria by which we may distinguish between them. It will be my endeavour to throw some light on these two points.

Now, the principal conditions with which I would deal are three :

(i) Poliomyelitis.

(ii) Acute myelitis.

(iii) Acute peripheral neuritis, or, as it is better called, acute toxic polyneuritis.

All of these may give rise to an acute flaccid paraplegia, with at first abolition of the tendon reflexes.

A word must first be said on the pathological side. Poliomyelitis is, as you all know, the disease which was formerly known as infantile paralysis; it is an acute inflammatory disease which may attack any part of the nervous system; the spinal cord, however, suffers most frequently and most intensely. On the disease in childhood I do not propose to dwell. A flaccid paraplegia of rapid onset in a child is, in the absence of bone or joint disease, almost certainly due to poliomyelitis. Less frequently it attacks adults and is then more likely to cause diagnostic difficulties.

* Read at the Æsculapean Society.

Anatomically the brunt of the disease falls on the anterior horns of the grey matter of the cord, hence the flaccid paraplegia.

The great majority of the cases of so-called *acute myelitis* are in reality syphilitic in origin. The process is a double one, a gummatous meningitis and a softening in the cord itself following thrombosis of the vessels from syphilitic endarteritis; usually it occurs within five years, often much earlier, after infection, thus affording a contrast to tabes dorsalis, which seldom occurs before ten years. Latterly the Wassermann reaction in the blood and the cerebro-spinal fluid, and the other changes which are known to occur in the cerebro-spinal fluid with the syphilitic process, have confirmed our previous conceptions as to the syphilitic origin of the vast majority of these cases.

The pathological basis of acute toxic polyneuritis is less certainly known. As it affects the lower limbs, it may be caused by alcohol—most commonly—by arsenic, or by the diphtheritic toxin. Other cases are of more doubtful nature, occurring sometimes after various infective disorders, especially influenza.

Now are there any clinical features which are characteristic of any one of these different conditions? There are in many cases. Take first poliomyelitis. The age of the patient is here a consideration of great importance. It is rare in any but children and quite young adults; uncommon over twenty, its incidence then rapidly diminishes.

The symptoms are often characteristic. The patient has usually been in perfectly good health. Then after a few days of slight constitutional disturbance, with generally some fever, the paralysis sets in more or less abruptly and rapidly reaches its maximum. Pain—contrary to what is usually stated in most text-books—is often severe, and tenderness of the limbs on manipulation generally quite a marked feature. Objective sensory disturbances in the shape of anæsthesia are, on the other and, conspicuously absent, and the sphincters of the bladder and rectum are very rarely affected. The limbs are quite flaccid, the deep reflexes—the knee- and ankle-jerks—abolished, and muscular wasting is rapid; loss of tone in the muscles is a marked feature. In any case of doubt the further course will generally clear up any difficulty. Recovery sets in, and the residual paralysis is always far less extensive than that present at the height of the disease. Usually the paralysis settles down into one or more groups of muscles, the remainder recovering their tone rapidly.

Mention, however, should be made of the so-called "jump cases" first described in this country by Sir Thos. Barlow. Here the disease, after originally involving one limb or the legs, suddenly progresses and attacks another segment of the cord, causing paralysis of arms or trunk.

To the case I have described the clinical picture of acute myelitis—using this term in the sense which I mentioned, namely, a syphilitic lesion—presents a clear contrast. The patient is older, generally over twenty-five at the least. After a few days during which some weakness of the legs may be felt or only sensations of pins and needles, numbness and tingling, paralysis sets in rapidly, and in the course of two to three days is usually complete. At first this paralysis is also a flaccid one, with abolition of the knee-jerks.

Later, and usually in the course of a week to ten days, the paralysis gradually becomes spastic. This is because the lesion is not a complete transverse lesion of the cord, the temporary flaccidity being due to the suddenness of the onset and the general vascular disturbance which is thereby occasioned. With the spasticity of the limbs go increase of the knee-jerks, the development of ankle clonus, and the appearance of a double extensor plantar response.

This alteration from a flaccid to a spastic paraplegia is never seen in poliomyelitis, and points to the greater involvement of the white as opposed to the grey matter of the cord. Sensory symptoms, again, furnish an important distinction. We have noticed that while pain may be a prominent and early symptom in poliomyelitis, objective loss of sensation is very rare. In acute syphilitic myelitis, on the other hand, pain is rare, while rapid and often complete loss of sensation is the rule; generally this loss involves all the ordinary forms of cutaneous sensibility—tactile, painful and thermal—and extends to a definite level corresponding to the situation of the lesion in the cord. Often the sensory loss is of the type known as "dissociated anæsthesia." This means simply that while some forms of sensation are lost, others are retained. When this occurs it is common to find that painful and thermal are more seriously

affected than tactile. The condition of the sphincters is another point of difference. In poliomyelitis it is rare for any bladder or rectal disturbances, except of a very temporary nature, to occur; in acute myelitis their functions are almost invariably severely attacked. The commonest state of affairs is retention of urine, leading to overflow if unrelieved, and obstinate constipation, which when relieved by an aperient is followed by temporary incontinence; in less favourable cases complete incontinence of both urine and fæces may be found. The final result again is usually quite distinct in the two conditions. Whereas in poliomyelitis the original and extensive paralysis clears up rapidly, leaving a residue in the shape of an atrophic paralysis of certain groups of muscles, in acute myelitis recovery—if adequate treatment has been employed—may be practically complete. Usually, however, a certain degree of spastic weakness of the limbs remains, accompanied by exaggerated knee-jerks and extensor responses. Sensation recovers before motion and control of the sphincters is commonly re-established. There are, therefore, numerous important points of distinction between the two conditions, which should usually be sufficient to ensure an accurate diagnosis.

The next class of case with which I wish to deal is peripheral neuritis or toxic polyneuritis. You are, of course, familiar with the two common forms of peripheral neuritis—lead palsy and the foot-drop of alcoholic neuritis. But occasionally this neuritis assumes a more extensive and acute character, so that a paraplegia may be rapidly produced, at first sight difficult perhaps to distinguish from an acute myelitis. The following points of distinction may be borne in mind.

(i) Sensory symptoms in polyneuritis are usually more marked and may vary from mere pins and needles to violent and agonising pains.

(ii) The degree of motor paralysis is seldom so complete and tends to involve the distal portions of the limbs more than the proximal.

(iii) While in the severer cases sensation is commonly lost, the anæsthesia does not partake of the segmental character seen in cases of acute myelitis—that is, absolute loss of sensation to a definite level—but is less complete and, like the motor weakness, involves the distal parts of the limbs more severely.

(iv) A very highly characteristic feature, particularly of the alcoholic cases, is often present, namely, hyperæsthesia of the affected limbs. Often patients cannot endure the legs to be manipulated at all, and I recently saw a case where the patient literally screamed with pain when attempts were made to remove his trousers.

(v) In polyneuritis there is, of course, no affection of the sphincters.

(vi) Rapid and severe muscular atrophy with the electrical reactions of degeneration occur.

(vii) Finally, the subsequent course of the case will amply serve to make a clear distinction, for in polyneuritis the paralysis does not, of course, become spastic, nor do the reflexes become exaggerated, nor the plantar responses extensor in type.

There is one symptom often present in cases of alcoholic neuritis upon which some stress may be laid; it is nystagmus. In some, to whom its appearance in this condition is unfamiliar, it often often causes doubt and difficulty, whereas it is a point of considerable value in confirming the diagnosis.

I have endeavoured briefly to lay before you an account of these three conditions which are certainly the most common causes of a rapid paraplegia in a person of previously good health. If I have dealt with too elementary points, I must crave your indulgence, but I have found that much confusion arises at times from a neglect of the simple facts of anatomy and physiology.

There are, of course, many conditions with which I have not dealt at all. Such events as the paraplegia of Pott's disease, or cerebro-spinal or other forms of meningitis, I do not propose to consider, nor such rare and difficult conditions as tumours of the spinal cord or of its membranes.

But there are two diseases in which it is perhaps not so widely known as it might be that a rapid paraplegia of a transitory kind may occur. These are tabes and disseminated sclerosis. Its occurrence in tabes is rare, but I have seen myself one well-marked example which it is worth while to relate.

A middle-aged man, who, after experiencing for some days a feeling of weakness in the legs, was quite suddenly seized with complete retention of urine, necessitating catheterisation. This was rapidly followed by practically complete paraplegia. On examination a flaccid paralysis of both legs, with abolition of the deep reflexes, was found. Characteristic changes in the pupils in the shape of

inequality, with a very sluggish reaction to light, however, were also found. At first sight the picture suggested a syphilitic myelitis, but the absence of any marked sensory loss corresponding to the extent of motor paralysis, rendered this diagnosis improbable. Further, the paralysis rapidly cleared up, without even becoming spastic, and in the course of about ten days there remained no doubt that the disease was tabes.

These cases of tabetic paraplegia usually, like those I have mentioned, recover in a short time; it is only by careful consideration of the case and paying strict attention to the symptoms and signs, such as pupillary changes or optic atrophy, that we can expect to arrive at a correct diagnosis.

Disseminated sclerosis is a disease which stalks under many guises, and it is only rarely that we see the classical case so well described by Charcot, with nystagmus, scanning speech, intention tremor, etc. One of the commonest varieties it assumes is the purely spinal form, a slowly progressive paraplegia. More rarely the onset is acute, and in these cases it is only by the subsequent course of the case and particularly by the remarkable and characteristic remissions which occur that a correct diagnosis is likely to be made. In an obscure and difficult case it may be useful to bear these two conditions in mind.

I have already, I expect, trespassed far upon your patience, and given what I am only too conscious is but a barren survey of a difficult subject. With prognosis and treatment I have not the time to deal adequately. In the discussion of this paper, however, I hope that any points either of prognosis or treatment will be raised; I shall be very glad to answer them to the best of my ability.

I have laid stress upon the pathological side and upon the diagnosis because it appears to me so essential to understand the former in order to arrive at the latter, and without an accurate diagnosis all prognosis and treatment are likely to produce results disappointing to doctor and patient alike.

February 1st, 1915.

Deep Cancers cured by X-Rays.—Bumm and Warnekros state that after disappointing extensive trials of mesothorium and other radio-active substances, they returned to röntgenotherapy, with a modified technique, and report two years of most encouraging results. The secret of success is to use hard tubes, by the cross-fire technique, to generate 500 X units in the depths. As about six-sevenths of the rays applied to the surface fail to penetrate to a depth of 10 cm., fully 3500 X units have to be applied to the skin to obtain 500 units in the focus. No serious injury results from this intensive exposure of the skin to the exclusively hard rays, even with 800 X units. At worst, the skin may blister, but this heals in three or four weeks, and a month of a transient local dermatitis is not to be considered when balanced against the cure of malignant disease. Seven cases of advanced cancer of uterine cervix, fourteen of the breast and one of the lung are reported, all showing remarkable benefit under the exposures, even the microscope confirming the complete retrogression of the cancer, as they show by slides of excised scraps. The tissues became indurated, with hyaline degeneration of the connective tissue, and there was not a trace of cancer by the fifth week in one typical case of a bleeding carcinoma in the uterine cervix, as large as a man's fist, the patient, æt. 37 years. The rays were applied externally alone, the distance from the focus 10 or 15 cm., the daily exposure totalling 60 or 80 minutes, the different fields of exposure overlapping so that each was exposed from 90 to 120 minutes. In two weeks the growth had subsided by one half, and by the end of the third week the retrogression continued spontaneously after the close of the course of treatment.

In one case the interval since treatment has been over a year, and the cure has persisted to date. The patient, a woman, æt. 42 years, was already in advanced cachexia with dyspnœa from an inoperable recurrence of cancer of the breast. The lymph-nodes in the axilla were much enlarged, both above and below the clavicle, and adherent. The patient returns every two or three weeks for a prophylactic exposure. One great advantage of Röntgen treatment over the knife is that any lymph-nodes, involved or other metastases, feel the curative influence of the rays on their way to the deeper focus. Ovarian cancers proved the hardest to influence, but good results were obtained in two cases with metastases left after pan-hysterectomy for ovarian cancer.

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We regret that owing to the war the number of pages must be diminished for a time.

TUBERCULOUS CERVICAL ADENITIS IN CHILDREN.

By JOHN FRASER, M.D., M.Ch., F.R.C.S.E.,
Assistant Surgeon, Royal Hospital for Sick Children, Edinburgh.

DURING the past few years one has had an opportunity of observing an unusually large number of cases of tuberculosis of the cervical glands: it may be of interest to summarise the impressions and ideas one has formed. The cases under review have been without exception operated on, and treated in the out-patient department of the Edinburgh Sick Children's Hospital, and if numbers are to be judged as authority for publication, one is justified, for the total number of cases operated on in the department during the past two years amounts to 300.

ÆTIOLOGY.

When one states that the infection of the gland with the tubercle bacillus is the actual ætiological factor, one is only providing the skeleton, so to speak; there are many details to be considered.

Age.—The material from which I have drawn my results has been limited to children below the age of twelve years; and the study of the age-incidence is instructive. During the first year of life there has been a larger proportion of cases than one would have expected. Tuberculosis is not common during this period and yet no less than 10 per cent. of one's cases could be classed in this group. This comparatively large incidence is due to the fact that these children were almost without exception bottle fed, no precautions having been taken to ensure the sterility of the milk. In the period which extends from one to two years the incidence was almost the same, a total occurrence of 27 cases or 9 per cent. During the period embraced by the years two to four there was a

striking increase in numbers. Between the ages of two and three there was a total of 50 cases; between the ages of three and four the number was 48. In other words a third of the total number of cases occurred during the periods of from two years to four. Beyond this point the number steadily diminished until in the last group, between the years of eleven and twelve, there were only 9 cases. The figures are accurately detailed in the accompanying table. There is a lesson to be drawn from the study of age-incidence. The comparatively high proportion of cases during the first four years of life is dependent on two factors, the imbibition of impure milk and the development of lymphoid tissue in the pharynx and naso-pharynx. The possibility of a milk infection probably explains the comparatively large proportion during the first two years. The high incidence during the second period is undoubtedly related to the development of lymphoid tissue, a tissue which, in its early formation, is highly absorptive, but not discriminative of or antagonistic to the absorbed products.

Table of Age-Incidence.

Years.	Number.
To 1 year	30
From 1 to 2	27
„ 2 to 3	50
„ 3 to 4	48
„ 4 to 5	21
„ 5 to 6	31
„ 6 to 7	28
„ 7 to 8	25
„ 8 to 9	19
„ 9 to 10	14
„ 10 to 11	15
„ 11 to 12	9

Type of bacillus.—Mitchell has recently carried out an investigation of the types of bacilli found in tuberculous cervical glands. He has found that cow's milk containing tubercle bacilli (bovine) is responsible in Edinburgh for no less than 90 per cent. of the cases of tuberculous cervical glands in infants and children. It is possible that the conditions of milk supply in the environment in which this investigation was made may be responsible for such a large proportion of bovine infection. None the less, bovine infection plays a more considerable part in the infection than has hitherto been suspected. Similar investigations on a less extensive scale have been carried out by others.

Park and Krumwiede examined a series of 54 cases; 27 of the cases were from children (5 to 16 years). In this group 19 were due to the human bacillus and 8 to the bovine organism. Oehlecker investigated 14 cases, 12 of which were children; of these 12 cases 8 were due to the human bacillus.

When the human bacillus is found to be the infecting agent, it is important to know the possibilities which have existed for the infection of the child from a "consumptive."

In Mitchell's series, a family history of pulmonary tuberculosis was found in three instances; in each of the three cases the human type of the tubercle bacillus was found to be present. It was interesting that in the bovine series, in no case was there a family history of pulmonary tuberculosis.

Portals of entry of the bacilli.—The great majority of tuberculous glands are infected by way of the lymph-stream, and the organisms gain admission in the area from which the lymph is drained. The common situations of entry are (1) the tonsil, (2) the lymphoid-tissue of the naso-pharynx (adenoids), (3) the teeth.

Of these three the tonsil is by far the most important. The organisms are carried into the crypts of the tonsil, being conveyed by dust or by such articles of diet as milk and cream. Their lodgment does not necessarily result in an actual pathological change, although a definite number of cases develop tuberculous tonsils (Stiles and Carmichael). The organism may pass through the tonsil into the lymph-stream, and the lymph which drains the tonsil finds its way into the upper set of the carotid glands.

Adenoid tissue and the tonsil of Luschka frequently harbour tubercle bacilli: Stiles and Carmichael found a proportion of 14 per cent. in fifty cases.* The distribution of the lymph from such sources is mainly to the chain of glands which lie along the posterior edge of the sterno-mastoid muscle.

The question of decayed teeth is often neglected when this matter is being discussed; the lymphatics from the teeth pass to the submaxillary group of lymphatic glands, and one has invariably found the

* These results included examination of the palatine tonsil.

disease of this group associated with decay in one or more teeth.

There are other rarer portals of entry. An apical phthisis may involve the supra-clavicular glands, and the infection spreads in an ascending course until it involves the higher chains of the neck. If the original infection has been an alimentary one the glands are usually infected through the medium of the blood-stream.

Routes of infection.—For all practical purposes one may exclude the possibility of direct infection; the organisms are carried to the glands by one of two routes, by the lymph-stream or by the blood-stream, the former is, of course, infinitely more common than the latter.

Predisposing infections.—In recording the clinical history of these cases one has been struck by the frequency with which the glandular involvement is preceded by the occurrence of one of the exanthemata and more especially measles. It would appear that the precursing disease so depresses the vitality that the gland readily falls a victim to the tuberculous infection subsequently infected or already in existence. It is sometimes stated that a gland which is enlarged as the result of a chronic irritation is in this way predisposed to subsequent true tuberculous infection: the truth of such a statement is doubtful. Looked at from the pathological aspect, one would expect the irritative changes to be resistant to any further infection.

Pathology.

Pathological anatomy.—Among the best descriptions of the arrangement of the cervical lymphatic glands is that given by Poirier & Cuneo. They divide the neck glands into two groups: a circular series and a vertical series. The circular series forms a chain around the base of the skull at the junction of the head and neck, and the constituents may be described in order from before backwards.

Almost in the middle line between the anterior bellies of the digastric muscles there lie the submental glands, they drain their lymph from the skin of the lower lip and chin and from the mucous membrane of the tip of the tongue and the anterior part of the floor of the mouth. Next in the series come the sub-lingual glands; they lie between the genio-glossi muscles upon the hyoglossus. The afferent lymphatics come from the under surface of the tongue and the floor of the mouth. The submaxillary glands lie in the digastric triangle between the mandible and the salivary gland. They receive their lymphatics from the anterior part of the face, including the inner half of the conjunctiva and the orbit, from the nose, from the mucous membrane of the mouth, including the teeth and gums, and from the anterior part of the tongue. Associated with this group there is the gland which lies on the outer surface of the mandible in relation to the facial artery—the facial gland. It draws its lymph from the side of the face, and it transmits it to the submaxillary group. Around the ear there are three sets of glands, the anterior auricular, the posterior auricular and the parotid. The anterior auricular group lie beneath the parotid fascia in front of the tragus, their lymph is derived from the root of the nose, the eyelids, the anterior part of the scalp and the anterior part of the external ear. The posterior auricular glands are usually only two in number. They are situated over the insertion of the sterno-mastoid into the mastoid temporal. Afferent lymph vessels pass to them from the posterior surface of the outer ear, and from a limited area of the scalp lying behind the level of the ear. The parotid lymphatic glands are buried in the substance of the parotid salivary gland; a small and special group of them form a short chain along the anterior border of the sterno-mastoid muscle and they have been given the name "superficial cervical" or "sub-auricular" glands. The parotid group receive lymph from the inner surface of the outer ear, from the middle ear, the nasal fossæ, the skin covering the outer thirds of the eyelids and from portions of scalp in the frontal, parietal, and temporal regions. Around the tip of the mastoid process a collection of glands is sometimes spoken of as the mastoid group. In this description they have been included in the group of superficial cervical glands. Last in the series come the occipital glands; they lie along the superior nuchal line, and their lymph is entirely derived from the posterior part of the scalp. The circular chain would be completed by mentioning the deep facial and the retro-pharyngeal glands, but as these are practically never affected with tuberculosis their description has not been included.

The vertical series may be divided into three chains, an anterior, a right lateral and a left lateral. The anterior chain is of little pathological impor-

tance, it is practically never infected with tuberculous disease; from above downwards it is composed of the anterior jugular, the pre-laryngeal, the pre-tracheal and the para-tracheal glands.

The lateral chains are the largest and the most important of all the cervical glands; each lateral chain is divided into an upper and a lower division by the anterior belly of the omo-hyoid muscle, and each division is again sub-divided into an anterior and a posterior group.

Of these four groups the upper anterior is the most important as it is the one most commonly affected with disease. It receives lymphatics from the glands of the circular chain lying in front of the ear, and in addition from the naso-pharynx including the pharyngeal and faucial tonsils. The lymph from the faucial tonsil is distributed to a special gland of the upper anterior group, and as this gland lies in the triangle formed by the posterior belly of the digastric, the common facial vein and the internal jugular vein, it is usually called the jugulo-digastric gland (Wood). The lower anterior group continues the anterior chain downwards as far as the mediastinal glands. The afferent lymphatics which enter these glands are derived from the larynx, trachea, œsophagus and thyroid gland; the group is also in direct lymphatic communication with the glands above.

The upper posterior group is situated behind the internal jugular vein upon the splenius cervicis and levator scapulæ muscles; the spinal accessory nerve runs through the centre of the glands. Wood has shown that the lymphatics from the pharyngeal tonsil enter these glands on their deep surface; there are also afferent lymphatics from the anterior chain, the posterior auricular, and the occipital glands.

The lower posterior group is a continuation of those above. They lie upon the levator scapulæ and scalenus medius muscles, behind the internal jugular. Below they become the supra-clavicular glands, and here they are divided into a superficial and a deep set by the omo-hyoid muscle and the middle layer of the deep cervical fascia. The afferent vessels are from the glands above.

Changes in individual glands.—When a healthy lymphatic gland is examined on section it is seen to have a somewhat kidney-shaped outline. There is a rounded convexity and a hilum in which the blood-vessels enter and leave the gland. The afferent lymphatics pierce the convexity of the gland while the efferent lymphatics pass out of the gland with the blood-vessels. The gland is encased in a capsule of fibrous tissue, and from the deep surface of the capsule septa of connective tissue pass inwards breaking up the gland into incomplete divisions. The interior is occupied by lymphoid tissue, but not entirely for between the lymphoid tissue and the fibrous capsule with its septa there runs a channel, bridged across by connective tissue. The channel contains fluid lymph. The scheme of the lymph-stream is that the flow enters by the afferent vessels at the convexity of the gland, it passes into the lymph-channel to circulate through the gland, it filters its way among the lymphoid tissue, and it leaves the gland by the efferent lymphatics from the hilum. The blood-supply to the gland enters by an artery at the hilum, and the vessel breaks up in the interior. There are comparatively few vessels in the capsule. A knowledge of these anatomical facts is essential to an understanding of the pathology.

An individual gland may be infected by way of the lymph-stream or by way of the blood-stream; as has been said, the former more commonly than the latter.

The situation of the primary lesion in the gland varies according to the route which the infection has followed. If the infection has been carried by the lymph-stream the first evidence of disease is found in the peripheral lymph-channel, because the organisms are here caught up and arrested by the crossing bands of connective tissue. A very different picture is presented when the infection is a blood borne one. The organisms are carried by the blood-stream into the centre of the gland and they become deposited at the point where the large vessel breaks up into a plexus of finer vessels. Therefore, if the diseased gland is examined sufficiently early, one is able to tell from the position of the original tubercle whether the infection has been lymphatic or hæmic.

The cut surface of a healthy lymphatic gland has a grey waxy appearance and to the naked eye the original tubercle presents the manifestation of a dull white point set in the grey background. Microscopical examination shows that the characteristic white appearance is due to the development of a tuberculous follicle. From the original deposit other follicles appear and the white point becomes

appreciably larger. Soon the pathological picture changes, caseation occurs in the centre of the original deposit, and macroscopically it becomes evident by a yellow appearance in the interior of the white patch.

Such changes occur simultaneously in a number of follicles and the individual points coalesce until the greater part of the interior of the gland is occupied by caseous material. Presently the central disease makes its way up to the capsule. While the central disease is developing, a certain degree of peri-adenitis almost necessarily occurs, and as this leads to a thickening of the capsule perforation of the latter by the disease may be delayed. When perforation eventually occurs it results in the formation of a peri-glandular cold abscess. The final stage is the perforation of the overlying fascia and skin by the abscess and the establishment of a sinus. The formation of a sinus almost necessarily means a mixed infection and pathologically this is evidenced by an increase in the degree and the extent of the peri-adenitis. There are some interesting and rather inexplicable points about the peri-adenitis of gland tuberculosis. For example, one constantly finds the gland and its capsule becoming adherent to the sterno-mastoid muscle and to the platysma, but it is rarely that one finds it attached to the digastric muscle. Fortunately, also, while the disease is often fixed to the sheath of the internal jugular, it is the exception to find it adherent to the vein itself.

One need only mention the microscopical characters; there is the development of a typical tuberculous follicle, and synchronus with its development there are changes in three other constituents of the gland—the connective tissue fibrils, the lymphoid tissue, and the endothelium lining the lymph-sinuses.

The connective tissue fibrils proliferate in number at the expense of the lymphoid tissue. In the more chronic varieties of tuberculous disease the proliferation may amount to a fibrosis and thus become the characteristic feature of the disease. The lymphoid tissue suffers from the proliferation of the connective tissue. If a part of a tuberculous gland uninfected with tuberculous disease be compared with the corresponding portion of a healthy gland one is at once struck by the comparative paucity of the lymphoid tissue in the former. The endothelial cells which line the lymph-sinuses proliferate and there is no doubt that they contribute in some measure to the epitheloid cells of the follicle. I have described the form of tuberculous disease which occurs in the large proportion of cases, the common caseating form. There are two somewhat unusual varieties, which one may term the lymphoid type and the fibrous type. The lymphoid type is apt to be mistaken for Hodgkin's disease. There is little tendency to caseation except in the late stages of the disease, the glands remain discrete, and there is little or no peri-adenitis. The lymphoid type is satisfactory from the operative point of view because the disease can be shelled out quickly, but in post-operative results it is unsatisfactory because of the tendency to recur. The distinctive feature of the disease is an excessive proliferation of the endothelial cells in the lymph sinuses.

Fibrous tuberculous disease one meets with more commonly in adults than in children. The glands are small and hard with usually some central caseation. It is a most undesirable type of the disease on account of the tendency which it has to the formation of dense adhesions with surrounding parts.

Groups of glands affected.—One may sub-divide the various collections of glands into three groups: the anterior deep cervical or tonsillar, the submaxillary, and the posterior cervical or sterno-mastoid. Of these three groups the one most commonly affected is the tonsillar; the disease has entered by the mucous membrane covering the tonsil and the faucial pillars and there may or may not be an associated enlargement of the tonsil. Secondary to disease of this group, the posterior chain almost inevitably becomes infected. It is very exceptional to find the submaxillary group becoming secondarily involved. The posterior cervical or sterno-mastoid group is, as has been said, most frequently an infection secondary to the tonsillar chain, but it may become primarily tuberculous as a result of infection from the naso-pharynx and posterior pharyngeal wall. The submaxillary group is poisoned by absorption of the bacilli from one or more decayed teeth. There are groups which are rarely affected and they only require to be mentioned. The pre-auricular or parotid is infected secondary to disease of the middle-ear. One has occasionally seen what appeared to be a primary tuberculous disease of the submental glands; it is

possible that the occipital glands may become diseased secondary to disease of the vertical chains.

Primary Groups affected.

Glands.	Number.
Anterior or tonsillar chain . . .	250
Posterior or sterno-mastoid chain . .	14
Submaxillary group	10
Parotid group	20
Submental group	4
Occipital group	2

The situation of abscess formation.—When caseation extends beyond the confines of the gland and becomes recognisable as a cold abscess there are certain situations in which the pus becomes most commonly superficial. Secondary to disease of the anterior group, the pus usually appears at the anterior border of the sterno-mastoid muscle and above the level of the omo-hyoid muscle. If the abscess has extended from tuberculosis of the posterior chain it becomes superficially apparent at the posterior border of the sterno-mastoid. Certain individual glands of the posterior chain sometimes lie behind the carotid sheath; caseation in this situation has a tendency to make its way inwards as a retro-pharyngeal abscess.

Clinical Features.

Clinical history.—The usual case history is characterised by the paucity of its symptoms. Often the one and the only thing to be mentioned is the fact that a swelling has appeared in the neck. The swelling may be apparently purely idiopathic, that is to say no precurrent condition can be held responsible for its development; more often, however, there are antecedent conditions which are frequently followed by the development of the disease. The commonest of these pre-current infections are the exanthemata more especially measles, and tonsillitis: the former act by diminishing the general vitality, the latter by lowering the local resistance of the gland. When the disease has established itself in the gland certain symptoms appear which depend on the absorption from the gland of toxic and deleterious products; the general appearance deteriorates, there is some loss in weight, a careful record of the temperature may show a slight evening rise, and the child tends to become somewhat cross and irritable in its disposition. In fact, one may, in a typical and simple case, classify the symptoms under three headings: introductory symptoms; glandular symptoms; and toxic symptoms.

It is sometimes the case that the symptoms are more severe than those which have been outlined. There are instances in which the disease is introduced with quite an acute onset; there is a history of a subacute tonsillitis, the cervical glands on one or both sides become painful and enlarged; the enlargement does not subside under the treatment usually adopted in a simple inflammation, but instead it passes on to abscess formation. It may be that it is only on the bacteriological examination of the pus that this type of disease is recognised as tuberculous. The explanation of this variety is either the introduction into the gland of a mixed infection of tubercle bacilli and the simple pyogenic organisms such as staphylococci and streptococci, or the infection of the gland with an exceptionally acute strain of the tubercle bacillus.

At the opposite extreme one finds an exceptionally mild form of the disease: that which is pathologically spoken of as the lymphoid type, and which clinically is characterised by an entire absence of symptoms beyond that of the enlargement of the gland. So far one has purposely avoided any mention of pain as a symptom of the disease. Usually it is negligible, but under two conditions it becomes evident: in the acute variety of the disease and in the chronic type when the pressure of a cold abscess is beginning to devitalise the skin.

Physical signs.—In the early stages of the disease an examination of the neck reveals an enlargement of the lymphatic glands in the usual anatomical situations. The enlarged glands are firm, discrete and painless. After some time the characters alter; the development of the disease in the interior of the gland has originated a peri-adenitis, and this becomes apparent to physical examination by the fact that the gland now becomes fixed either to surrounding structures or to other glands. Associating pathology and physical signs still further, the central caseation becomes sub-capsular and this is evidenced to physical examination by a localised softening when the gland is submitted to firm palpation. Cold abscess formation is next observed and pathologically it means an eruption

of the caseous material through the diseased capsule. It is interesting to note the suddenness with which this last sign may appear; it may be the matter of a day, even of a few hours, and the sudden increase in swelling is often the first thing to alarm the patient or his friends. With the development of the cold abscess the peri-adenitis increases; it becomes impossible to delimit the outlines of the underlying gland, and the edges merge imperceptibly into the surrounding parts.

The abscess continues to increase in size and as a result of the pressure which it exerts, the skin becomes irritated and inflamed. The sequel is that the skin gives way and the caseous *débris* escapes on to the surface. It is inevitable that the edges of the sinus become infected with tuberculosis, and a tuberculous ulcer is thus formed.

Diagnosis.

Actual diagnosis.—One knows that a glandular enlargement is tuberculous by certain distinctive features. The age of the patient is important; in children there are comparatively few diseases which produce a glandular enlargement, and of such tuberculosis is the commonest. The second feature is the slow and insidious development of the disease, with the comparative dearth of general features. An absence of pain is another reliable feature, and finally one would mention caseation and cold abscess formation.

A glandular enlargement characterised by such features is very unlikely to be anything else than tuberculous.

Differential diagnosis.—It has been said that there are comparatively few conditions in children which one is apt to confuse with glandular tuberculosis; there are, however, one or two which must be mentioned.

(1) Simple sub-acute adenitis. The mistake lies in confusing this condition with the acute type of tuberculosis, and a distinction may be exceedingly difficult. The points in favour of a simple infection are the occurrence of a previous or coincident throat infection, the considerable degree of pain, the presence of fever and the improvement of the condition under simple treatment. If dubiety still exists and abscess formation has occurred the uncertainty is cleared up by withdrawing some of the fluid with a fine needle and submitting it to microscopical investigation.

(2) Simple chronic glandular hypertrophy. Children suffering from irritative conditions, such as scalp impetigo, tonsils and adenoids, etc., are liable to develop an enlargement of the glands draining the diseased areas. Difficulty may arise in excluding this condition from the milder varieties of tuberculous disease. Its distinction hinges on two points: it does not progress, and when the source of irritation is removed the glandular swelling disappears.

(3) Hodgkin's disease. This may be distinguished by the distinctive absence of peri-adenitis, the discreteness of the glands, the multiplicity of the enlargement and, if any other proof is needed, by the absence of softening and abscess formation.

It is unnecessary to discuss the differential diagnosis of such diseases as lympho-sarcoma and the glandular enlargement of lymphatic leukæmia; they are very rarely met with in children.

Prognosis.

Glandular disease is one of the least dangerous of the many forms of tuberculous disease. It may spread from gland to gland throughout the neck, but it is exceptional to find a general secondary dissemination, or an invasion of neighbouring organs such as the lungs. Therefore, speaking generally, it may be taken that the prognosis as regards eventual cure is distinctly good. Under three conditions the prognosis is somewhat guarded; these may be classified under the headings of age, position, and multiplicity of the lesion.

Age.—The younger the child when the disease appears, the more serious does the prognosis become; the first two years of life are especially serious. The majority of cases at this period run a rapid course, the glands enlarge quickly, they caseate rapidly and chain after chain become diseased.

Position.—Disease of the upper glands is less dangerous than disease of the lower. In the latter situation there is a tendency for the disease to extend downwards where it may ultimately involve the lung. Disease of the posterior chain is more dangerous than disease of the anterior group, for the reason that the posterior infection may become complicated by a retro-pharyngeal abscess.

Multiplicity of the lesion.—The coincident or subsequent infection of a number of different groups

of glands is a serious development as it indicates a considerable want of resistance to the tuberculous infection.

Treatment.

The treatment may be considered under the sub-headings of general treatment, local conservative treatment, and operative treatment.

General treatment.— The principles guiding general treatment are too well known to require recitation; they are attention to hygiene and diet. Whatever be the line of local treatment adopted, the demands of general treatment must never be neglected. It may happen that in the very early stages of the disease general treatment may be sufficient to abort the condition.

Local conservative treatment.—(1) The use of hot fomentations. In every case in which the enlargement of the gland is complicated by peri-adenitis the local conservative treatment should be begun with the use of frequently changed hot fomentations. Peri-adenitis is demonstrated by some degree of fixation of the gland, the presence of overlying tenderness, and an indistinctness of the gland outline. These features all disappear when a thorough course of hot fomentations is carried out, and whether the after-treatment is to be a continuation of the conservative or an operative one, either course ought to be introduced by a preliminary fomentation treatment.

(2) Trypsin injection. Dr. Wilhelm Baetzner, of Berlin, first introduced local trypsin injection in the treatment of surgical tuberculosis. The method is applicable in the treatment of tuberculous cervical glands, irrespective of whether abscess formation has occurred or not. Baetzner recommends the preparation produced by Fairchild Brothers and Forster, New York. The injections are sold in glass ampoules holding 1 c.c. and the trypsin is dissolved in 60 per cent. glycerine. For use in the treatment of gland tuberculosis the solution ought to be diluted with 10 volumes of physiological salt solution. The injection is given subcutaneously in amounts of ½ to 1 c.c. at intervals of six to eight days. The solution acts by producing a stimulation of granulation tissue and a subsequent fibrosis and shrinkage of the gland. Certain undesirable symptoms may develop as a result of the injection. Locally there may be swelling, redness and pain and generally there may be evidences of fever, malaise and albuminuria. These complicating features very quickly disappear, but in view of their possible occurrence, it is well to keep the patient under observation for some days subsequent to the injection.

(3) Congestive treatment. A modification of Bier's congestive treatment may be used in the treatment of tuberculous glands. Klapp's cups are applied over the mass and a vacuum is maintained for periods of one hour twice a day. It is not uncommon that a cold abscess develops while the treatment is in progress. In such a contingency the abscess is opened and the treatment continued, every precaution being taken to ensure the sterility of the process. This method of treatment possesses few advantages to recommend it. It is of necessity a prolonged one, the tendency to the development of cold abscesses is considerable, and the scars which result from their incision are often multiple and unsightly.

(4) Local skin applications. A multiplicity of these have been at one time or another introduced. They vary between such applications as tincture of iodine, producing the subsequent improvement by an inflammatory reaction, and special drugs like allyl-sulphide, which is supposed to produce an improvement by its specific action on the tubercle bacillus. No local application has yet been introduced for which a curative effect can be claimed.

(5) George P. Muller has recently drawn attention to the value of X-rays in the treatment of early tuberculous cervical glands. Applications are given at intervals of ten days and such applications last from five to ten minutes.

(*To be concluded.*)

February 8th, 1915.

Suicide with Potassium Permanganate.— Adler reports the case of a woman, æt. 37 years, who took 10 grm. of potassium permanganate in solid form and died on the fourth day. When seen an hour after she had taken the drug the breathing was very irregular, with pauses up to twenty seconds, during which the pulse dropped from 72 to 44, but the heart action kept good. Apnœa has not been mentioned before as a symptom. It subsided with the signs of acute intoxication, and was due, as in the two other cases on record, to the corroding action of the chemical on the throat entailing phlegmons in the neck and pneumonia. —*Journ. A.M.A.*, vol. lxiii, p. 1511.

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We regret that owing to the war the number of pages must be diminished for a time.

TUBERCULOUS CERVICAL ADENITIS IN CHILDREN.

By JOHN FRASER, M.D., M.Ch., F.R.C.S.E.,

Assistant Surgeon, Royal Hospital for Sick Children, Edinburgh.

(*Concluded from page* 48.)

Tuberculin treatment and serum treatment.—In the administration of tuberculin, one of two possible methods may be followed: the tuberculin is given in steadily increasing doses producing a general reaction, or it is maintained at a uniform small dose sufficient to produce a simple focal reaction. In the treatment of tuberculous glands it is the latter desideratum which is required and the injections therefore consist of small doses which are not increased in amount. In regard to the type of tuberculin which should be used, success may be obtained with any variety if its characteristics and dosage are thoroughly understood. In view of Mitchell's results showing a large proportion of gland tuberculosis which owes its origin to infection with the bovine bacillus, the question arises whether the tuberculin administered should be human, bovine, or a mixture of both. Some believe in the use of autonomous vaccines and treat a lesion due to a bovine bacillus with a bovine tuberculin and *vice versa*; others use a human tuberculin for conditions due to the bovine bacillus and *vice versa*. Good results have been obtained by both methods and the probability is that human and bovine bacilli are so closely related that the respective tuberculins produce very similar results.

A number of different sera are now on the market for the treatment of tuberculosis: it is sufficient to mention Marmorek's, Spengler's and Mehnarto's. The author has had recent experience with Mehnarto's serum, the dosage for a child is 2 c.c. and at the end of a week 4 c.c. are given and the injections are maintained at this amount

and repeated twice a week. The results are promising.

Operative treatment.—Indications for operation. Some authorities have contended that the certainty that enlarged glands in the neck are tuberculous is sufficient to demand operation and excision; an opposition exist who claim that in practically no instance is operation called for. It is wiser to approach the subject with an open mind and to recognise certain well-defined indications for and against operation. One would summarise the indications for operation as follows:

(1) When the disease is distinct yet limited an early operation with a small incision may prevent a spread to other chains of glands.

(2) When the disease in the gland has passed on to caseation.

There are other subsidiary indications, but these are the evident and pressing ones. There are very definite contra-indications.

(1) Age. One hesitates to recommend operation in children under the age of two years. If the disease has developed at this early age it is very probably extensive in distribution and unless the operation is going to guarantee a complete removal it is better not attempted.

(2) Absence of caseation. As long as softening is not appreciable in the gland there is a possibility of the disease subsiding under conservative measures.

(3) The extent of the disease. A disease so extensive that a complete removal cannot be guaranteed ought to be accepted as a contra-indication to operation.

(4) The character of the diseased gland. Under this heading one more especially means the degree of peri-adenitis which is present and the occurrence of sinuses. In the fibrous type of the disease associated with extreme fixation of the glands and complicated with sinuses it is probably wise to adopt conservative means.

Each case must be decided on its own merits in the question for or against operation.

Preliminaries to operation.—It is important to pay careful preliminary attention to the hygiene of the mouth. In the course of the operation it may be necessary to cut into the parotid and infection not infrequently spreads from a septic oral cavity along Stenson's duct into the parotid gland and thence into the wound. The local preparation must include the shaving of the scalp on the affected side. Failure to observe this detail may easily lead to a post-operative infection of the wound. The decision of the question of the anæsthetic is a most important preliminary. The ideal anæsthetic would be one in which the bleeding would be reduced to a minimum and in which the proximity of the anæsthetist to the operator could be prevented. The risks of high spinal anæsthesia are too great to justify its employment. Intravenous anæsthesia with ether, hedonal or paraldehyde fulfil the advantage of not having the anæsthetist in immediate proximity to the operator, but this advantage is more than counterbalanced by the excessive bleeding with which these anæsthetics are associated, not to mention the increased danger to life which they entail when compared with methods in more common use. In most cases one's choice is limited to ether, chloroform, or a mixture such as A.C.E. Chloroform has the advantage of being associated with less bleeding than either ether or a mixture; it is distinctly more dangerous, however. The excessive bleeding of ether may be considerably reduced by arranging that during the operation the head is kept elevated: this position does not materially increase the difficulty of the operation. The author uses a mixture of equal parts of ether and chloroform; the anæsthesia is primarily induced by ethyl-chloride (2 c.c. for a child) and continued with the mixture.

Operations.

Operation for removal of tuberculous submaxillary glands (Fig. 1).—The head is placed with a pillow below the nape of neck and the face turned towards the opposite side. The incision begins in the middle line of the neck midway between the symphisis menti and the hyoid bone, it extends downwards in the middle line to the level of the hyoid bone, it then passes outwards across the neck to the anterior border of the sterno-mastoid muscle and upwards along the anterior border of the muscle to end opposite the angle of the jaw. The flap of skin thus outlined is dissected upwards until the body of the jaw is exposed. At the anterior border of the masseter muscle the facial artery and vein are seen: they are divided at this point between ligatures. The reflection of the flap exposes the submaxillary salivary gland; the lymphatic glands

lie around it. The glands thus exposed may be described as lying in a quadrilateral space: above the body of the lower jaw forms the boundary, in front there lies the anterior belly of the digastric, below the posterior belly of the same muscle, posteriorly the boundary is completed by the anterior border of the sterno-mastoid muscle and at a deeper level by the stylo-maxillary ligament. Lying over the glands the facial vein is seen: it is ligatured and divided before it crosses the digastric muscle. The glands are separated from the structures which form the floor of the space, from before backwards they are the mylo-hyoid muscle, the hyo-glossus muscle and the superior constrictor of the pharynx. The separation is carried out by passing the finger upwards on the deep surface of the glands. As this dissection is being completed the facial artery is again exposed as it crosses behind the digastric muscle to disappear beneath the glands.

The artery is divided between ligatures. The whole blood-supply to the glands is now controlled and the remaining dissection may be completed. The glands are dissected away from before backwards; a portion of gland tissue passes with the duct of the salivary gland beneath the posterior edge of the mylo-hyoid muscle; the duct is clamped and divided. As the glands are being dissected off the surface of the hyo-glossus care must be taken to avoid injury to the structures which lie upon it, more especially the hypoglossal nerve. When the removal is complete the skin flap is restored to its position and drainage is secured through a special opening made as a stab-wound at a level just above the wound.

Operation for removal of tuberculous glands from

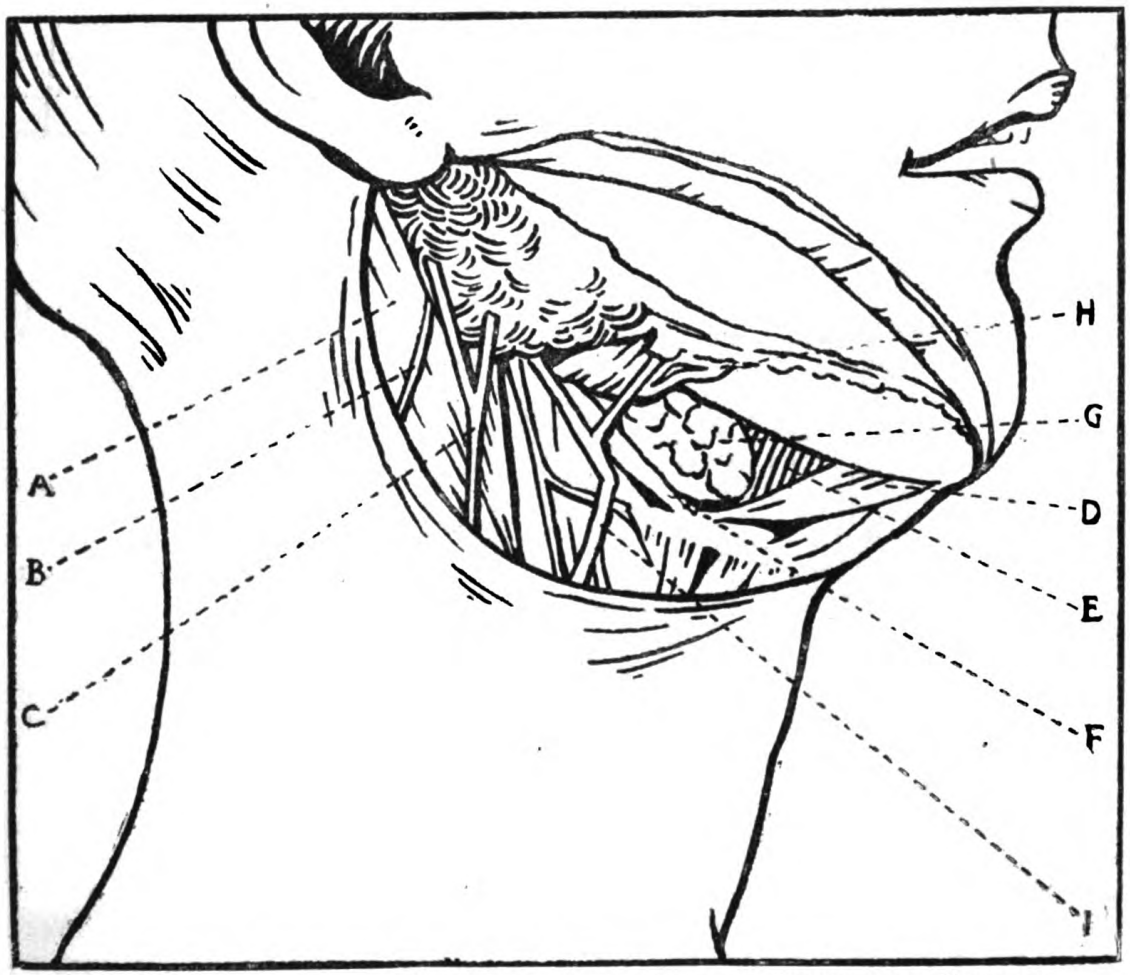

FIG. 1.—DISSECTION FOR THE REMOVAL OF GLANDS FROM THE SUBMAXILLARY TRIANGLE.

A. Sterno-mastoid muscle. B. Great auricular nerve. C. External jugular vein. D. Digastric muscle, anterior belly. E. Mylo-hyoid muscle. F. Posterior belly of digastric muscle. G. Submaxillary gland. H. Facial artery. I. Common facial vein.

the anterior and posterior triangles (Fig. 2).—The position of the head is similar to that described in the previous operation except that the chin is tilted upwards so as to open up the complete side of the neck. The type of incision varies; the oblique incision extending from the tip of the mastoid process forwards and downwards has long been a favourite. Stiles recommends a ∩-shaped incision when the disease is extensive and involving both triangles. The incision begins at the side of the body of the hyoid bone, it passes upwards to behind the angle of the jaw, it crosses the sterno-mastoid muscle immediately below the mastoid process and it passes downwards along the anterior border of the trapezius muscle as far as the upper border of the clavicle.

I have recently employed a transverse incision: it passes across the neck almost transversely lying in a natural crease. It gives very good access and it leaves an unnoticeable scar; it extends from the anterior edge of the trapezius muscle to the middle line of the neck. The skin edges are freely reflected and a liberal separation is essential to a satisfactory operation. With the reflection of the skin certain anatomical structures stand up; the sterno-mastoid muscle passes obliquely across the wound: behind it lies the posterior triangle uncovered to any extent by platysma except in its lower part, but probably occupied by enlarged glands if the disease is in any way extensive. Appearing at the posterior border of the muscle there are certain superficial nerves; the great auricular appears at the middle of the posterior border of the muscle and passes upwards towards the mastoid process; the transverse superficial cervical appears at the posterior border of the muscle and passes forwards across the neck; the supra-clavicular nerves from the same superficial origin extend downwards towards the clavicle. Passing vertically down the side of the neck there is the external jugular vein, extending from the space between the angle of the jaw and mastoid process above to the centre of the clavicle below. The vein is clamped in the lower part of the wound and

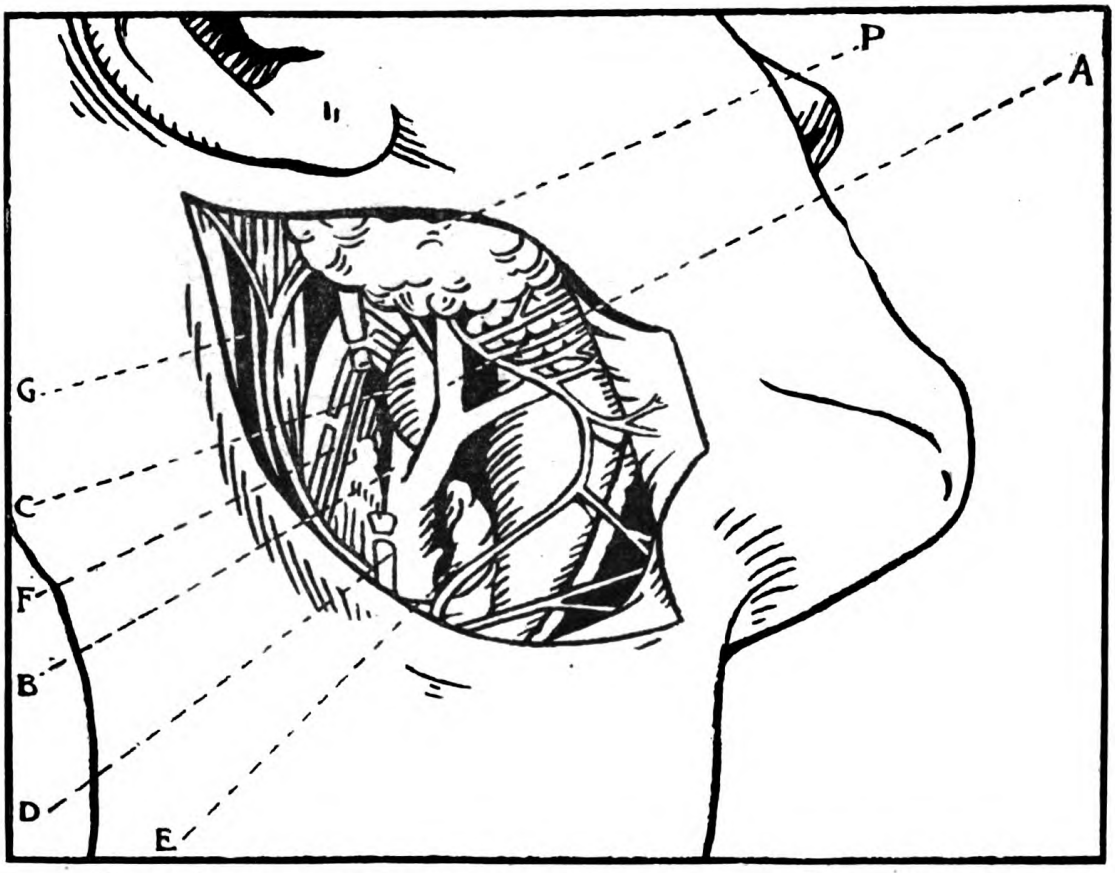

FIG. 2.—DISSECTION FOR THE REMOVAL OF GLANDS FROM THE CAROTID TRIANGLE.

P. Parotid gland. G. Sterno-mastoid muscle. C. Tonsillar gland. F. Spinal accessory nerve with superior sterno-mastoid artery. B. Internal jugular vein. D. External jugular vein. A. Common facial vein. E. Anterior deep cervical glands.

separated upwards. As the vein approaches the upper end of the sterno-mastoid it is surrounded by a small chain of superficial glands—the superficial cervical. These are frequently diseased and they are removed with the vein. In front of the sterno-mastoid muscle the anterior triangle is covered over with a thick layer of platysma, which is bulged forwards by the enlarged deeper-lying glands. The glands are removed first from the anterior triangle; the platysma is reflected by a T-shaped incision, the top of the T runs along the anterior border of the sterno-mastoid muscle, and the limb passes from the centre of this incision forwards to beneath the chin. The platysma is thus reflected in two flaps which can be replaced afterwards when the edges of the wound are restored. The division of the platysma exposes the enlarged glands. They may be said to lie in a muscular triangle bounded above by the posterior belly of the digastric, in front by the edge of the depressor muscles of the hyoid bone, and posteriorly by the anterior edge of the sterno-mastoid muscle. The anterior edge of the sterno-mastoid is defined and the definition carried sufficiently deeply to expose the spinal accessory nerve accompanied by the superior sterno-mastoid artery, a branch of the occipital, both passing obliquely downwards and backwards to enter the deep surface of the muscle. The glands are separated above from the submaxillary salivary gland and at a deeper level from the posterior belly of the digastric muscle. In front they are detached from the anterior belly of the omo-hyoid muscle and from the hyoid bone. The removal is completed by separating the glands from their deep relations, from the common facial vein, the middle and inferior constrictors with the thyro-hyoid membrane, and more posteriorly the carotid sheath with the included vessels. It is quite exceptional to see such structures as the superior thyroid and lingual vessels, etc., and therefore they have not been included in the description. The glands at their highest point lie in relation to the parotid gland, and a portion of this latter structure has frequently to be removed with the diseased glands.

The removal of glands from the posterior triangle is a simpler operation than the anterior dissection. With the separation of the skin the superficial nerves already mentioned are seen, but more important than the superficial nerves, one has to recognise the spinal accessory, which after it leaves the posterior border of the muscle becomes at first almost subcutaneous. The nerve is followed for some distance and cleared from the surrounding tissues. In removing the glands from the deeper relations of the posterior triangle the author begins by retracting the sterno-mastoid muscle and separating the glands from the posterior margin of the sheath of the vein; this is done from the anterior border of the muscle. In this position the glands may be said to be divided into a small group above the spinal accessory nerve and a larger chain below. When the separation from the vein sheath is complete the sterno-mastoid is allowed to slip forwards and it is held aside in that position. The glands are now dissected off the muscles which form the floor of the posterior triangle, especially the splenius capitis and the levator scapulæ. Care should be taken to follow out portions of the glandular chain which pass between the two above-mentioned muscles. The separation of the glands on their deep surface completes the dissection. It is often unnecessary to retract the sterno-mastoid forwards; if the glands are small the dissection can easily be made from the front, but if they are of any size more room is obtained from the posterior position. In closing the skin-wound it is advisable to suture the platysma back into position, and if it has been reflected in the way described the stitching is not difficult. Catgut is used and the observance of this detail prevents subsequent depression of the scar. The skin edges are brought together with clips or stitches, the former for preference; drainage is established through a special stab-wound.

Complications during operation.—Considering these in the order in which they might occur, one would first mention the presence of an abscess. This can scarcely be called a complication, but in the eyes of some it constitutes one. When it is met with one pays no attention to it; if by chance it is opened the pus will not produce a subsequent infection of the wound. The entire abscess wall is removed with the deeper underlying glands. If the abscess should lie below the skin care is taken to avoid having the incision passing through devitalised tissue, and therefore the skin-wound is planed to lie either above or below the abscess.

Injury to the jugular vein.—The dangers of this complication have been over-rated; the danger of

air embolism is more apparent than real. If the opening in the vein is a small one it may be possible to repair it by a fine silk suture; it is not advisable to apply a lateral ligature as there is the ever present danger of it slipping. When the opening in the vein is large it is better to proceed at once to ligature the vein above and below the injured spot. It may be necessary to apply an additional ligature to the common facial vein as it enters the main vessel.

Injury to the spinal-accessory nerve.—This complication occurs for two reasons: it may be unavoidable as when there are masses of adherent glands around the nerve, or it may arise as a result of the fault of not recognising the nerve early in the dissection and therefore cutting it later by mistake. Early recognition will largely help to prevent the complication. If the nerve is damaged an attempt should be made to re-suture it with fine silk thread.

Adhesion of the glands to the carotid sheath.—If it is recognised that the glands are densely adherent to the carotid sheath it is not advisable to persist in attempts at their separation. It is better to open the sheath low down in the neck, and, differentiating its structures from one another, to ligature the vein between two ligatures, taking care to avoid inclusion of the vagus nerve. The vein having been divided between the ligatures it, with the afferent glands and the remains of the sheath, is dissected upwards. On reaching the junction of the common facial vein two ligatures are again applied and the intervening vein divided. The separation is continued upwards to beyond the limits of the disease, and at this point the vein is again secured and divided.

Late complications; recurrence.—No complication has brought the operation into greater disrepute than this one. A thorough removal of the disease is the best way to avoid its occurrence, but occasionally it appears in spite of every precaution. There are certain situations in which recurrence notably occurs. The first is the parotid; during ordinary dissection it is usually impossible to detect the presence of diseased glands in this situation unless they are appreciably enlarged, recurrence, therefore, is often unavoidable. The second situation in which a recurrence may develop is in the posterior part of the neck between the adjacent edges of the splenius and the levator scapulæ muscles. This occurrence may be avoided by a complete dissection at the time of operation.

February 8th, 1915.

Tubal Pregnancy due to Tubal Tuberculosis.—C. Mannheimer and E. Bovin point out that not infrequently does gonorrhœal salpingitis lead to extra-uterine pregnancy. That this may also occur as a sequel to tuberculous salpingitis is shown by the following cases:

A nullipara, æt. 30 years, had been married eighteen months. Several years earlier she had been treated for tuberculosis of the apex of one lung, from which she recovered. Menstruation had previously been regular, scanty and painless. Early in February menstruation was succeeded by pain in the abdomen and back, and by slight hæmorrhage. When it had lasted a fortnight she was examined. The urine was normal, and there were no signs of active disease in the lungs. The uterus was anteflexed, movable, and not enlarged. The neighbouring structures seemed normal. As the patient suffered from pain and giddiness after the examination, she was admitted to hospital where rest in bed arrested the hæmorrhage. She still suffered from occasional violent pain in the right lower abdomen, and the temperature was slightly raised. Gradually a tender, resistant swelling developed to the right of, and behind the uterus, increasing from the size of a plum to that of a hen's egg. In the middle of February an attack of violent pain was accompanied by pallor and a weak and rapid pulse. The pouch of Douglas was found to be distended. Laparotomy was at once performed, and a considerable quantity of fluid blood was found in the abdominal cavity. In the pouch of Douglas a partially organised blood-clot, of the size of a hen's egg, was found adherent to a coil of small intestine. The right Fallopian tube was distended to the dimensions of a thumb, and its distal end was open. The right ovary, which contained a small corpus luteum, was normal. The left tube was also distended, and when a probe was introduced into its distal end it collapsed, yielding fluid blood. The left ovary was normal. Only the right tube was removed, the left tube and both the ovaries being left. Convalescence was delayed by bronchitis, but when the patient was ultimately discharged the pelvic organs seemed normal on palpation. Microscopic examination of the right Fallopian tube showed acute miliary tuberculosis of its mucous membrane. A few chorionic villa were found, but otherwise there was no sign of a fertilisation of an ovum. The fluid blood found in

the left Fallopian tube had probably been absorbed from the abdominal cavity. It is also probable that this tube was tuberculous, and it is, therefore, a moot point whether, if the tuberculous nature of the condition had been recognised at the operation, the left tube should have been removed or not. Tuberculosis of the Fallopian tubes may heal spontaneously, and the patient being a young married nullipara, an operation involving sterility was most undesirable. On the other hand, owing to the left Fallopian tube remaining in the pelvis, the patient ran the risk of a left tubal pregnancy.

A nullipara, æt. 34 years, who had been married nine years, and had suffered from spondylitis in childhood, had previously suffered from no disease of the reproductive organs. Early in December she began to suffer from attacks of vomiting and pain in the lower abdomen. When she was admitted to hospital a month later, the uterus was found somewhat enlarged and displaced to the left and forwards by a tumour, as large as a goose's egg, to the right of and behind the uterus. Laparotomy showed the tumour to consist of the much distended, right Fallopian tube, which was surrounded by several clots of blood. The condition of the left tube was not recorded. The excised right tube contained a 4 cm. long fœtus, and was lined by a spongy, greyish-white mucous membrane containing many tubercles.

If, as is probable, tuberculosis of the Fallopian tube is a relatively common cause of extra-uterine pregnancy, the question is raised whether, in every case of operation for extra-uterine pregnancy, both Fallopian tubes should be removed on the assumption that both tubes are diseased. At all events, the operator should bear in mind the possibility of the extra-uterine pregnancy being due to tuberculosis.—*Hygiea*, p. 134.

Treatment of Tetanus by Injections of Carbolic Acid.—The large number of cases of tetanus during the present war has induced Talamon and Pommay to direct attention to the value of subcutaneous injections of carbolic acid. This method has the advantages of being practically free from danger, easily carried out in the most primitive hospital, and of insignificant cost. The solution employed differs from that originally devised by Baccelli and consists of 10 grm. of crystallised glacial carbolic acid, 50 grm. of pure glycerine and distilled water to 1000 grm., the whole to be sterilised. As soon as the diagnosis is made 20 c.c. of this solution is injected into the subcutaneous cellular tissue every four hours. Each dose contains 20 cgrm. of carbolic acid, so that the daily quantity administered in twenty-four hours is 1·20 grm. of phenol, an amount which appears to be absolutely free from danger. The injection is made on the outer side of the thigh or on the anterior abdominal wall. An ordinary glass hypodermic syringe which has been sterilised by boiling is all that is required. The site of injection should be cleaned with a tampon of wool dipped in ether or alcohol, but it is well to avoid tincture of iodine. Care must be taken to penetrate into the subcutaneous tissue to avoid superficial sloughing. In the majority of cases there is but slight pain at the site of injection, but occasionally the skin becomes reddened and in one case an abscess formed. The discomfort of these inflammatory symptoms usually yields to the application of a moist dressing. Apart from the characteristic darkening of the urine, the writers have observed no toxic symptom. The daily dose of 1·20 grm. in six injections was usually continued for two to three weeks, and was followed by half that amount for eight or ten days till the rigidity finally disappeared. With the injections chloral was administered in doses varying from 6 to 8 grm. daily by the mouth, or when this was impossible, owing to trismus, by the rectum. On one occasion a rash resembling measles was observed which was attributed to the chloral. It rapidly disappeared.

Of six patients thus treated five recovered, the fatal case being of exceptional severity. All the patients were civilians and in good health till the receipt of the injury or onset of tetanus, but the writers consider this method as specially suitable to tetanus of moderate severity complicating the injuries of war. If by the third day the temperature has not fallen to normal amputation is indicated; otherwise generalised infection and rapid death from septicæmia is inevitable. Fortunately, in cases treated as above described, this desperate condition is rare. Only three such cases had been seen by the writer, two of which recovered after amputation, while the third, in which amputation was not performed, was fatal.—*La Presse Médicale*, p. 684.

THE BRAIN IN HEALTH AND DISEASE. By JOSEPH SHAW BOLTON, M.D., D.Sc.Lond., F.R.C.P.Lond. Professor of Mental Diseases, Leeds University. London: Edward Arnold. Price 18*s.* net.

The title may possibly suggest a text-book for the alienist, but such is not the case. The theme is cerebral physiology and pathology, not discussed like some of the later books on bio-chemistry and bacteriology, but a return to the question of cerebral function on the now well-established basis of anatomy and physiology. The first half of the book deals with cerebral function in the normal brain, the second with that function in the abnormal organ, or rather the insane brain. Under the latter come the subjects of amentia and dementia, which are considered in detail, with an abundance of clinical types to illustrate the various features and grades. Dr. Bolton has the faculty of facile expression with ready illustration, as evidenced in such chapters as the "Human Cerebral Function" and "Paranoia." As indicated, the book is meant for the general student, and it is certain to appeal deservedly to a large circle of readers. About a hundred illustrations are provided, as well as an excellent index.

INFECTION AND RESISTANCE. By HANS ZINSSER, M.D., Professor of Bacteriology at the College of Physicians and Surgeons, Columbia University, New York. London: The Macmillan Co. Price 15*s.* net.

The work is intended primarily for the medical student. We fear that the author has over-estimated the cerebral capacity of that individual, for the book is more adapted to the practitioner or the laboratory worker. The title is sufficiently elastic to embrace, as indeed it does, the whole field of bacteriology. Immunity—natural, acquired, or artificial—is dealt with at much length, especially the methods of conferring the active form. In the so-called incubation period of infectious disease, two portions are distinguished—one necessary for the growth, distribution, and accumulation of the bacteria, the other the time necessary for the action of the toxin which may be secreted, but the incubation time of the toxin is evidently a subject which has not, so far, been entirely investigated.

Infectious diseases are described as the result of a struggle between two variable factors—the pathogenic powers of the bacteria on the one hand, d the resistance of the subject on the other, each of these again modified by variations in the conditions under which the struggle takes place. The whole subject is presented with clearness and simplicity.

THE BRITISH PHARMACOPŒIA OF 1914.

The year 1915 is responsible for the introduction of a new pharmacopœia. Whether or not the new copy will be regarded as an improvement on 1898 is a question which might be variously answered both by the medical man and by the pharmacist. The book is not handled or studied by the profession to any degree, for we wonder, indeed, on how many book-shelves it finds a place. The link between the medical man and the British Pharmacopœia is largely posological at present, though it is quite possible that under the Insurance Act the component part of the general prescription must be found between the covers of this official book.

The chief features of the new edition which should engage the close attention of the prescriber are as follows:

Tincture of strophanthus and aconite have been increased in strength, and the dose now is 2–5 minims. Laudanum follows suit, so that 20 drops of the 1898 tincture are represented by 15 in the new issue.

A new hypodermic injection of strychnia appears, and its dose, like the other official injections, is 5–10 minims.

Likewise a new salt of iron appears, viz., sacharrated phosphate, the dose 5–10 gr.; the sacharrated carbonate is retained, the dose 10–30 gr. Phosphorus pill is half the old strength, spirit of juniper double. Vinegar of squills is double the old strength with half the previous dose. The following preparations have been altered in strength, but have the same dose as before, viz., solution of perchloride of mercury, tincture of belladonna compound camphor, digitalis nux vomica, and ammoniated opium. Colchicum tincture is half the previous dose. Carbolic acid lozenges are half the previous strength.

In regard to external applications, belladonna plaster is half the former strength, mercury liniment and the ointments of carbolic acid, mercury, ammoniated mercury, compound mercury, are all reduced, while calomel ointment is increased.

The scruple is practically omitted, while ʒj is suggested for liquids only, grains being used when solids are indicated.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE

ON

GUNSHOT WOUNDS.*

By Major W. MCADAM ECCLES, M.S., F.R.C.S., R.A.M.C., T.F.,

Surgeon to St. Bartholomew's Hospital.

GENTLEMEN,—When I went away North at the end of July to the meeting of the British Medical Association I never thought that, in the middle of November, I should be giving you a clinical lecture on gunshot wounds, and for two reasons. Although we had dreaded this war, and, as you know, this Hospital, in 1909, arranged, at the suggestion of Sir Alfred Keogh, a Territorial General Hospital, which is now in Camberwell, containing 520 beds, we hardly thought it would really come. Also because my knowledge of gunshot wounds was then exceedingly small, being limited to those which have occurred in connection with civil practice in this Hospital.

I shall necessarily have to be very brief and fragmentary in my remarks on account of time.

As we are dealing to-day chiefly with gunshot wounds, a rough, but fairly accurate, history of projectiles from small arms may be interesting. From 1700 to 1840, for practically 150 years, the small, smooth, muzzle-loaders were used, and the projectile was a spherical bullet of soft lead. A similar lead bullet is still used at the present time, as we shall see, but not in the small arms, it comes from the artillery shrapnel. From 1851 to 1864 percussion-cap muzzle-loaders were used, and for them the bullet was cylindro-conical, and was still of soft lead. Then came a considerable advance in the breech-loader (1864–97), with two types of bullet—the ovoid, which was a curious bullet, and one not often seen, even in museums, at the present day, and the cylindro-conical bullet of

* Delivered at St. Bartholomew's Hospital.

lead, or a mixture of lead and some other metal to make it harder

Then the advance from 1891 to the present day, the day of the small calibre magazine rifle, was very great. This rifle fires a cylindro-conical bullet of hard lead with a cupro-nickel casing.

I throw on the screen specimens of British, German, French, and Belgian rifle bullets and some round shrapnel bullets. Three of the bullets are sharp pointed, the German bullet very sharply pointed. The Belgian is not nearly so pointed (Fig. 1).

I now pass to a very interesting point, namely, the regional distribution of gunshot wounds. This table gives the percentage fairly accurately of the cases we have had at St. Bartholomew's section of the 1st London General Hospital. Notice that the extremities come first, with 71 per cent., the upper limb and lower limb being about equally often wounded. The head and neck show 10·6 per cent.; the thorax 8·2 per cent.; the back 5·35 per cent. Wounds in the back are not received while running away, but are due in most instances to shrapnel falling from a height on a man while he is lying down. The abdomen and pelvis give 4·8 per cent. The abdomen and pelvis show such a small percentage in this series, because we do not get bad cases of this type of injury over here; they are probably more numerous in the base hospitals in Northern France. I have ventured to make a rough classification of the wounds we have seen here. First of all there are the wounds in which the projectile perforates the tissues, so that there is a wound of entrance and a wound of exit, and the damage that has occurred in the transit of the projectile through the tissues has to be dealt with. This kind of wound is generally caused by sharp-pointed rifle bullets. But a very large number of the wounds are non-perforating, and these are the more difficult to classify. There are generally lacerated wounds, and they are produced by the round bullets, shrapnel, and shell fragments, but sometimes even by the sharp-nosed bullets.

I have classified these as:

(1) The superficial or "brush-burn" wound, where the bullet has just grazed the skin itself, sometimes for a considerable distance. We had one man with such a wound, 7½ in. long.

(2) A deeper wound, the "trough" wound, and this is often seen on a finger (Figs. 2 and 3).

English. French. Belgian. German.

FIG. 1.—Types of bullets.

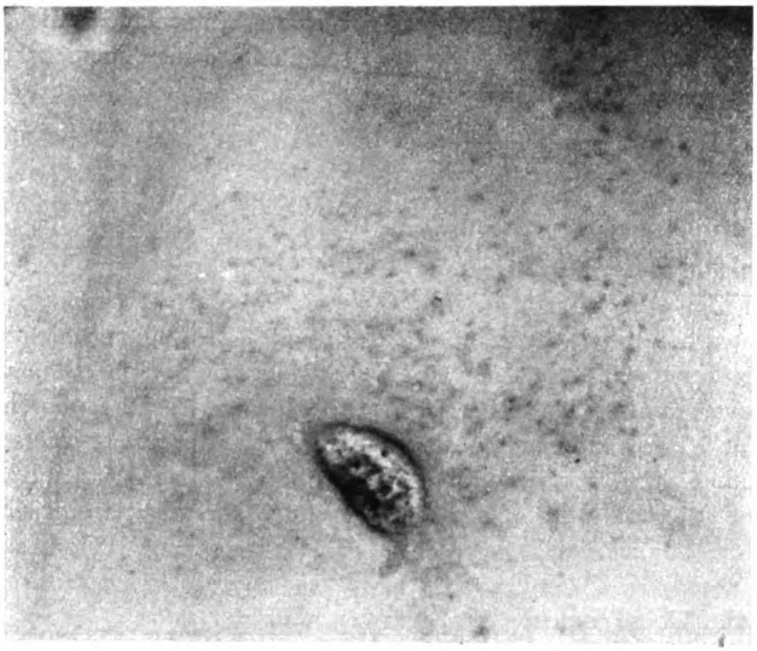

FIG. 2.—"Trough" wound of back, surrounded by a large number of pustules.

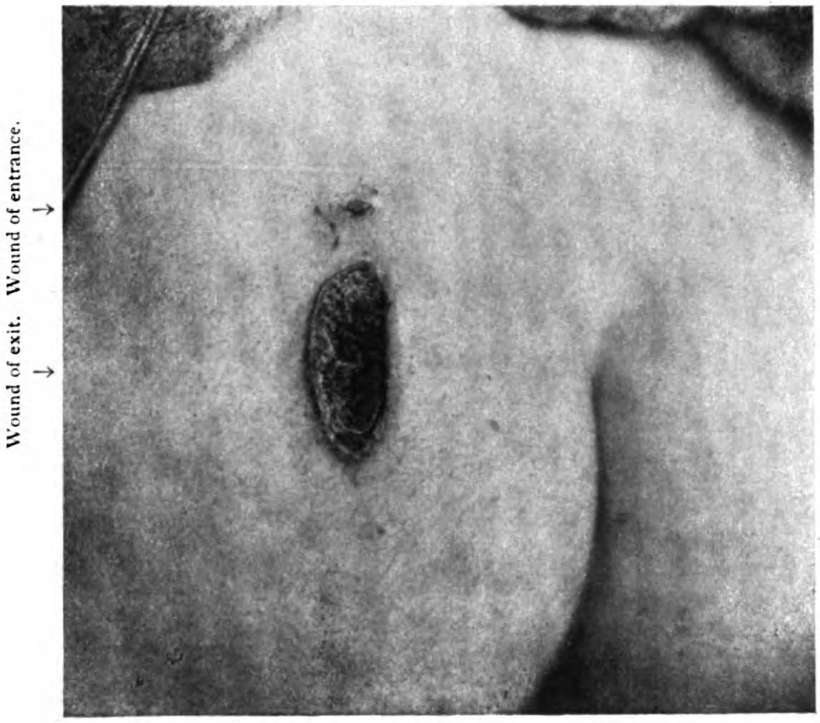

FIG. 3.—"Trough" wound of left buttock, caused by a rifle bullet. This wound took five weeks to heal.

(3) A much deeper lacerated wound, the broad "pit" wound (Fig. 4).

(4) The punctured wound, the narrow "pit" wound.

(5) The "total," in which the part is literally blown away.

This first wound (Fig. 2) is of a "trough" wound on the back, made by a piece of shrapnel, not by a bullet. I am glad to show you this, because all round the wound there are a number of little pustules, a condition with which many men have been coming over to us. There has been the terrific fighting at Ypres, and many of these minor superficial wounds have not been dressed more than once, and a good deal of pus has collected under the dressing; bacteria have been sown round about the wound, and a crop of pustules has resulted. These quickly disappear with antiseptic treatment.

Another photograph (Fig. 3) shows exceedingly well a superficial perforating wound. It was in a man who was lying down, and the bullet came from the front and hit him in the buttock. The aperture of entrance is less than the diameter of a threepenny piece. The bullet then passed under the skin for a short distance and made the wound which you see in its exit. There have been a great many of these wounds, most of them caused by the sharp-nosed German Mauser. A large number of the deeper perforating wounds are not septic and heal rapidly, and the man is very soon well and fit to go back.

This photograph (Fig. 4) shows the deeper "pit" wound. I cannot show on this print the wound of entrance which I showed you on a previous photograph. The wound of exit is clearly seen, as well as the immense pit leading to the bone, and the severe laceration. The wound just involved the knee-joint, and a mild septic condition resulted, for which an incision has had to be made on the outer side. He has done remarkably well.

Figs. 5, 6, and 7 illustrate varieties of "pit" wounds.

"Trough" wounds have been exceedingly frequent, especially in fingers and toes. As a man is firing his rifle, a bullet comes and makes a trough across the side of his finger, and the diagram shows the trough going down to the side of a phalanx.

Some skiagrams will illustrate the presence of bullets in the tissues, bullets which have not per-

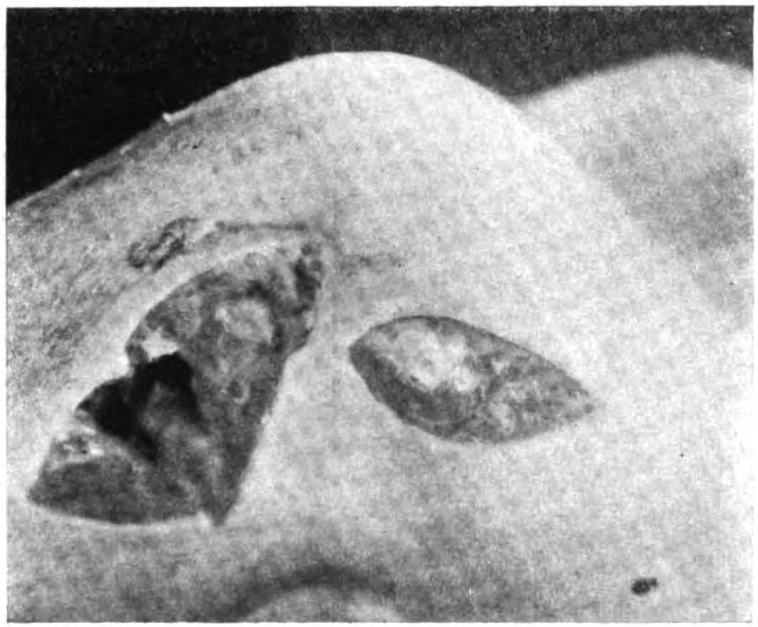

FIG. 4.—Deep "pit" wound of the outer side of left leg just below the knee, caused by a sharp-nosed bullet, which entered on the *inner* side of the leg, fractured the tibia, and carried the fragments of bone out through the soft tissues on the outer side. This wound was thought by some, but erroneously, to have been made by a "dum-dum." The limb was saved, but with a stiff joint.

FIG. 5.—"Pit" wound of the front of the left forearm, made by the semicircular fragment of the time-fuse of a shell, shown lying above the wound.

FIG. 6.—Deep "pit" wound of right elbow, caused by a large fragment of shell striking the back of the elbow and shattering lower end of humerus and upper ends of radius and ulna. Recovery with movement at the site of elbow-joint.

forated (Figs. 8 and 9). A Mauser bullet (Fig. 8) entered a hand on the radial side, near the second metacarpo-phalangeal joint. It traversed the palm of the hand and did not come out. Notice that the bullet, which we presume entered with its sharp end first, has turned round, and its

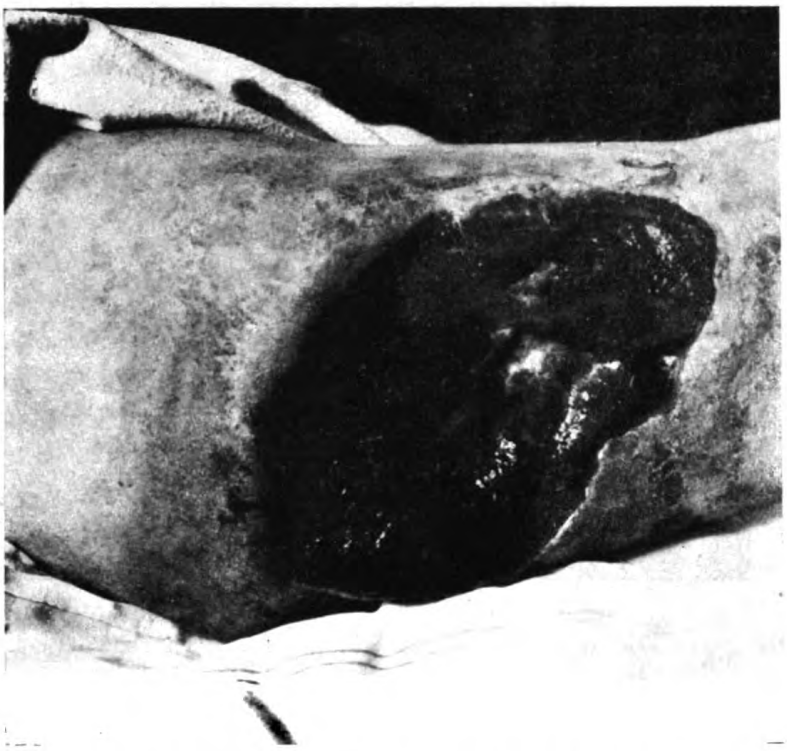

FIG. 7.—Deep "pit" wound on the back of the right thigh, caused by a sharp-nosed bullet striking a ration-tin in the overcoat pocket.

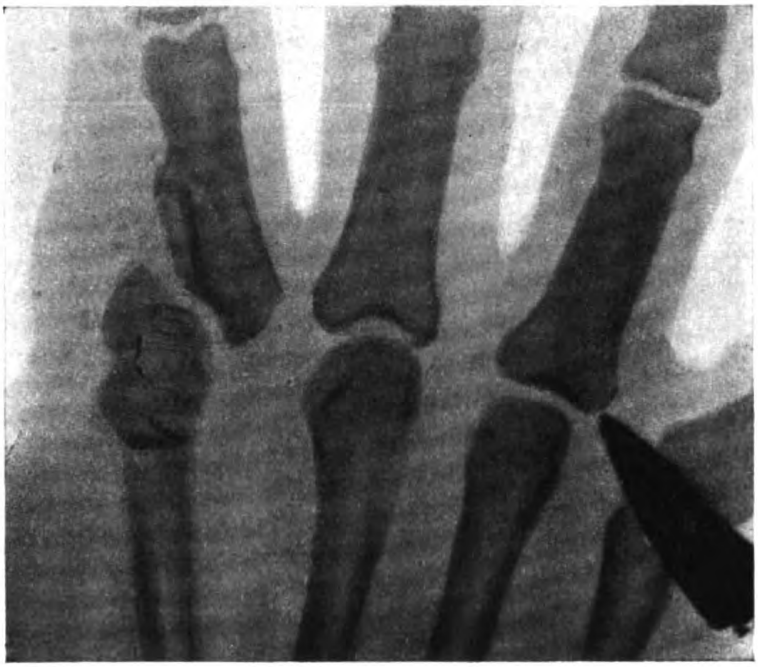

FIG. 8.—Sharp-nosed bullet which entered the hand on the radial side, fractured the base of the proximal phalanx of the index finger, and then passed across the hand and turned on its long axis. The wound of entrance was very small.

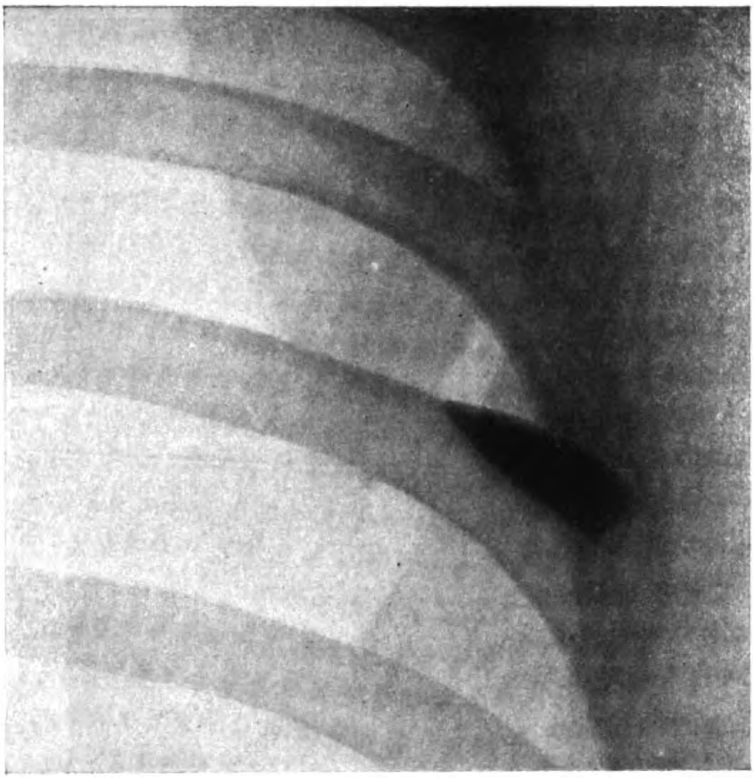

FIG. 9.—Sharp-nosed bullet in the tissues of the thoracic wall.

point faces the wound of entrance. We have had a number of cases in which a bullet has apparently done this. It is a little difficult of explanation. It may be, of course, that the bullet in these cases enters by its blunt end. That, however, I do not think was the case, because the appearance of the wound of entry does not seem to indicate it. Other skiagrams show the presence of the round shrapnel bullet. A man was lying down firing, and a shrapnel bullet entered his right scapular region, and instead of remaining there, it travelled right across and was extracted from the region of the opposite scapula. Yet, between the two wounds there was apparently no indication of any pain or trouble. In fact, the man had no idea that the bullet had gone across to the other side, and it was not until a skiagram was taken that this very obvious round bullet was discovered on the opposite side. Here is a round bullet in the foot. Notice the marked difference between the shadow of the bone and that of the bullet. The bullet is in the sole of the foot, and has not touched the bone.

I shall now pass on to speak of some of the dangers of these wounds. First of all, let us consider the danger to vital organs. Such wounds are not seen very often in the case of the soldiers who come here, because the men who are shot through a vital organ usually die quickly. But there have been a number of very interesting cases where the bullet seems to have gone through vital parts, and yet the men have survived, particularly of wounds across the thorax. This is a stereoscopic skiagram of a man's chest, and it shows a Mauser bullet to be definitely in the hilum of the right lung, a fairly vital part, and one in which a bullet should cause symptoms. But this man did not even know he had it in his chest, and the symptoms were so slight that the bullet has been left there. The radiogram was taken a fortnight after the bullet entered, so very little harm seems to have occurred as there was no septic material taken in with it.

Perhaps the most remarkable case was that of a man who was, on November 4th, struck just internal to the coracoid process of the left scapula by a Mauser bullet (Fig. 10). He walked one and a half miles after he was wounded. When admitted here on November 6th, he was found to have a marked left hæmothorax, but no apparent alteration in the heart's action, except that its rate was ninety-six per minute. A careful X-ray screen examination showed the bullet in the muscular tissue, with the sharp end pointing forwards, and oscillating with each beat of the heart. (Full details of this case will be published later.)

I now pass to the question of hæmorrhage. We are apt to think that injuries to the larger vessels

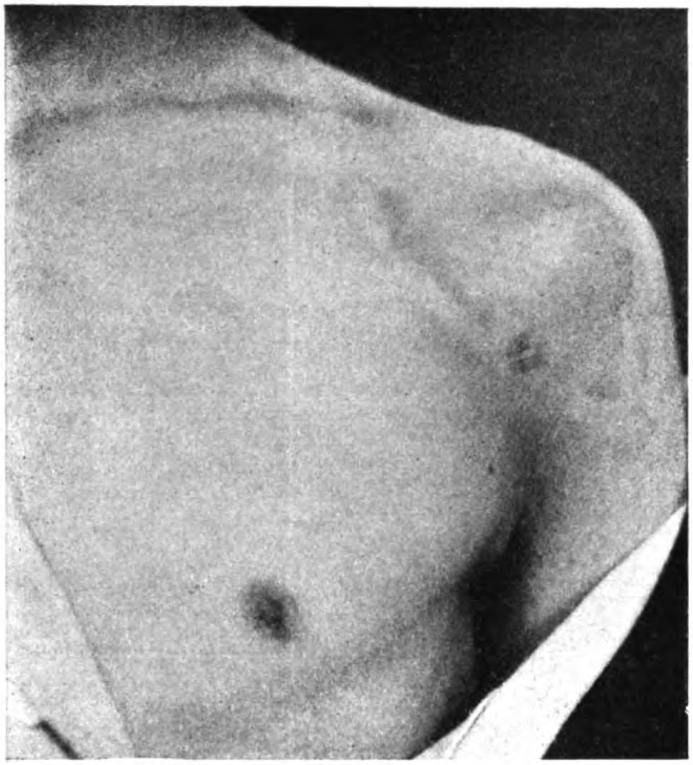

FIG. 10.—Wound of entrance of a sharp-nosed bullet, which, after penetrating the lung, lodged in the muscular tissue of the posterior aspect of the left ventricle on November 4th, 1914. The man is alive and retains the bullet.

must necessarily be fatal. But this is not always so, and there has been several cases which prove the contrary. A man came complaining that he had had a shrapnel wound of his left thigh. A skiagram demonstrated a piece of shrapnel in his thigh. The question always arises, when there is a piece of shrapnel in a somewhat deep position, as to whether it is better to leave it alone, or whether to carry out an extensive dissection to cut it out, because firstly, the localisation by stereoscopic views does not always make it easy to bring about removal; and secondly, a wound which was aseptic may become septic, as the result of the disturbance caused by the attempted removal of the foreign body. But in this case the piece was so accurately localised that we considered it could be easily removed. It was 1½ in. deep from the inner and lateral aspect of the middle thigh, and obviously behind the femur. The wound of entrance was just over the mid-point of Hunter's canal. We cut down over the wound of entrance, because it was septic and required cleansing. When Hunter's canal was reached, gentle spunging caused furious hæmorrhage to occur, evidently from a large artery. Temporary compression of the femoral on the proximal side stopped the hæmorrhage, and we were then able to see, at the bottom of the wound, a clot which had come out of a very jagged wound of the superficial femoral artery. A little further examination demonstrated that the wound of the artery was not merely an anterior surperficial wound, but the cut went right through the artery, the vessel having been perforated by the piece of shrapnel. We ligatured the artery on the proximal and distal sides of the lacerated wound, and then went deeper, and found the shrapnel lying posterior to the artery itself. The patient has done well, and no further secondary hæmorrhage has occurred.

One other case of secondary hæmorrhage may be recorded. A fairly smart hæmorrhage took place without warning from a bayonet wound in the upper arm, the blood coming from the cephalic vein, not from an artery. Although he bled profusely at the time, and got very frightened—much more frightened, he said, than when he received the wound—he has recovered without a further attack.

(*To be concluded.*)

February 22nd, 1915.

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE ON GUNSHOT WOUNDS.*

By Major W. MCADAM ECCLES, M.S., F.R.C.S., R.A.M.C., T.F.,

Surgeon to St. Bartholomew's Hospital.

(*Concluded from p.* 64.)

Now I come to speak of fractures. They are, naturally, very common in these gunshot injuries, because bone resists, and the bullet, although not being itself explosive or expansile, has, when it strikes or goes through bone, an expanding effect. For this reason, hard tissues, such as bone, splinter and become, as it were, ruptured in a large number of directions.

Here is a skiagram (Fig. 11) of a man who had a very extensive injury to the lower part of his right upper arm, and you will notice that his humerus has been fractured very badly. I particularly want you to notice that this sharp point of bone is just the region where the musculo-spiral nerve comes to the front, for that nerve was caught over that. Although it was an open—what used to be called a "compound"—and a septic fracture, it has united with the formation of a large amount of callus, and I think he will have an arm which he can use, though not in warfare.

Here is another very instructive fracture of the humerus (Fig. 12). It is from shrapnel, and there is a round bullet, and some pieces of another bullet which has spluttered, together with a comminuted fracture of the humerus. It is necessarily an open fracture, but fortunately, unlike the other case, no septic material was carried into the wound.

Nerve injuries, unfortunately, are very common in these gunshot wounds. Bullets, when they strike a nerve, damage it, either by concussion or dividing it, or a nerve may get caught in callus about a fracture. In the case of the fracture of

* Delivered at St. Bartholomew's Hospital.

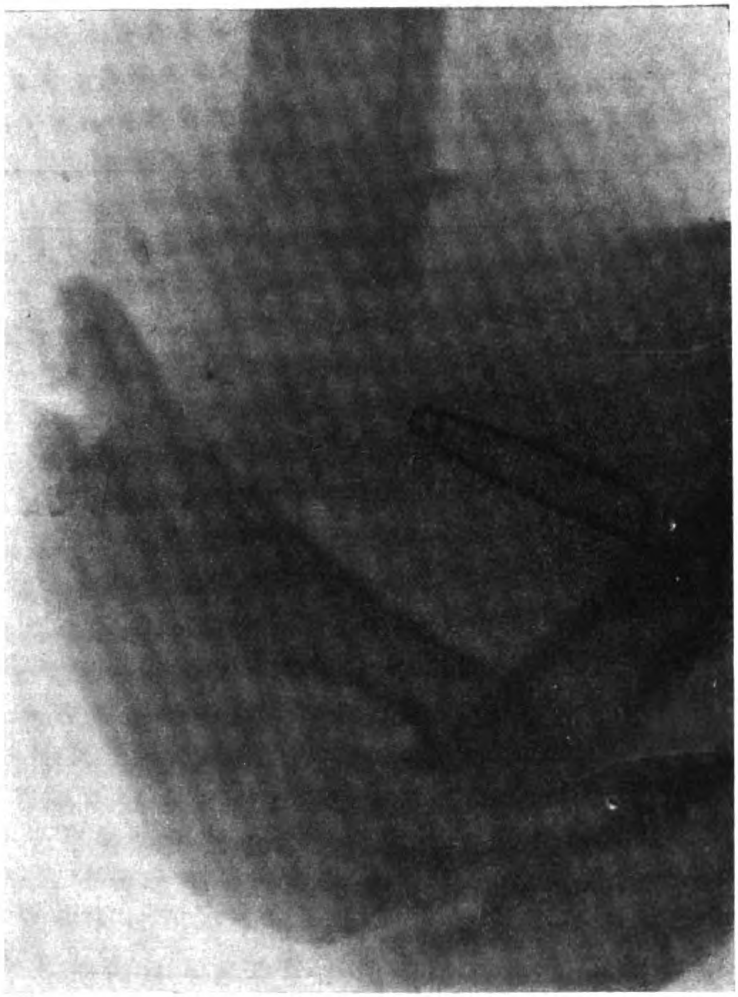

FIG. 11.—Open comminuted fracture of the right humerus.

the humerus I have mentioned, the man came in with tremendous sepsis, and was very ill indeed; he could not sleep, and he had an excruciating pain along the ulnar side of the dorsal aspect of his forefinger, so that even morphia, used fairly liberally, did not help him greatly. We were unable to operate at once because of the septicity. Fortunately, however, by care and treatment, the sepsis became less, and the nerve was cut down upon. We found the musculo-spiral nerve hitched over a sharp point, and evidently his severe neuralgia was due to the irritation of the sensory part of the musculo-spiral nerve. He also had wrist-drop, but that he recovered from, and he has entirely lost his pain. This was a typical injury to the peripheral nervous system. Now two cases of injury to the central nervous system; they were both in the lumbar region. Take this skiagram of a round shrapnel bullet (Fig. 13). It went in between the third and fourth lumbar vertebræ, smashing up, to some extent, the arches of both, and it was lying in the muscles by the side of the spine. But in addition, the spinal canal had been opened, and we are afraid that the thecal sheath itself was involved: he had a slight loss of power in his sphincters for a time. An unfortunate feature of these shrapnel wounds is that practically all of them are septic, and this man, since the removal of that bullet, has been pouring out pus. Fortunately, if there was a wound in the thecal sheath it has been closed, so there is no spinal meningitis.

The next case (Fig. 14) is likewise interesting, implicating the second lumbar vertebra. He came in with diminished knee-jerks, inability to pass his water, overflow incontinence, and some incontinence of fæces. His paraplegia was not complete, and he had some sensation in both lower limbs.

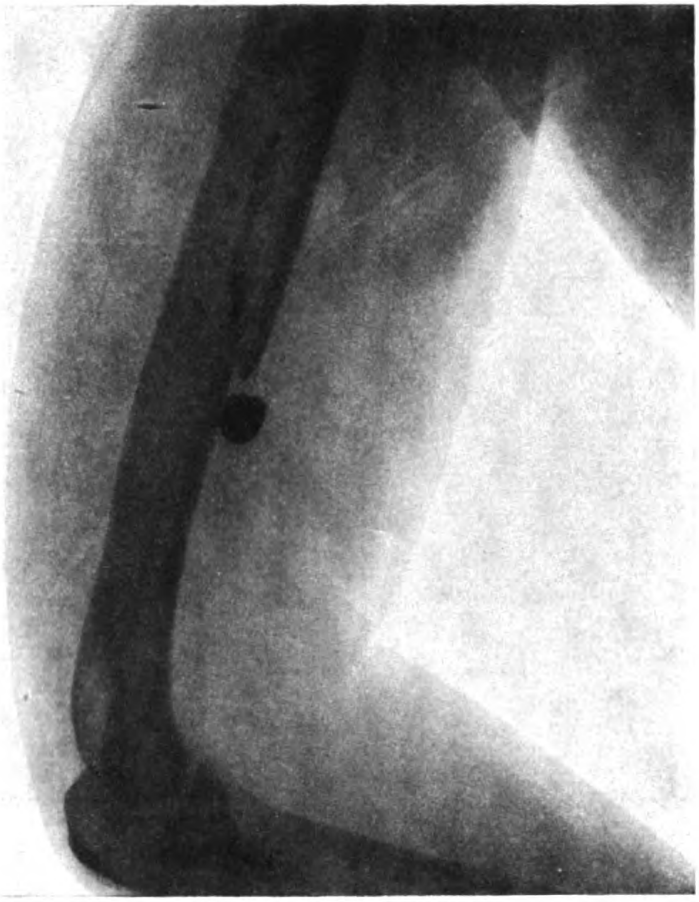

FIG. 12.—Spiral fracture of the left humerus with shrapnel bullets in position. Union and healing without suppuration.

As his wound was septic we waited for a few days to try and get it cleaner. After this an operation was performed, and the larger piece of shrapnel removed. It was lying amongst the cords of the cauda equina, and it looked as though one or two of the strands had been divided by the shrapnel missile. He improved and his urine became very much better than it was, and he began to get more power over his limbs, but he died of septic meningitis.

The question of sepsis in relation to gunshot wounds is very important. All these wounds are exceeding liable to be infected. There are two reasons for this. One is that the man in many of these cases is necessarily none too clean. He cannot help it: it is one of the "minor horrors" of war. And the second reason is that these projectiles may carry in septic material in the shape of clothing and other articles which may be superficial to the skin. All of you know that the bacteriology of the wounds in the present war, so far as they have involved the men of the Allied armies in France, has been of very considerable interest, because two types of organisms have, at any rate in the latter part of August and practically the whole of September, been very prominent, namely, anaërobic bacteria and the tetanus bacillus. A number of explanations have been given for this. One, which is very probable, is, that it is due to the extraordinary amount of cultivation which has taken place in the parts of France about the Marne and the Aisne Rivers, where so many wounds have been inflicted. In other words, there has been much manuring of the land, and these anaërobes live to a large extent in excreta. So that the septicity of the wounds has been extraordinarily frequent, and the number of cases of tetanus in the early stages of the war great. Fortunately, both these bacteria seem latterly to be on the wane. Whether it be that the men are taking more care, or whether they are now in trenches in parts where these bacteria are not so plentiful, it is difficult to say, but the emphysematous gangrene cases are fewer in number.

Unfortunately, I have not time to enter upon the discussion of the treatment of these wounds, but I may be able to do so in a subsequent clinical lecture.

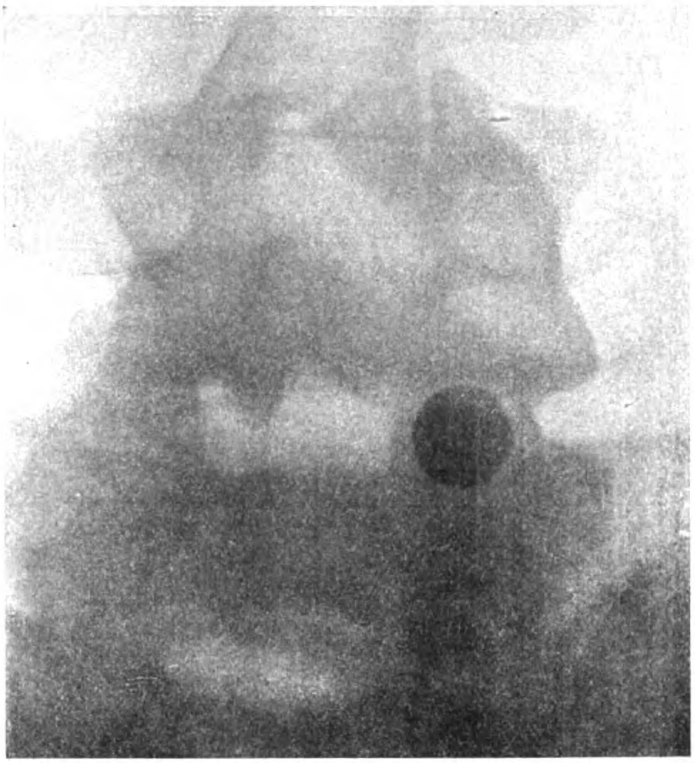

FIG. 13.—Round shrapnel bullet in intervertebral disc between fourth and fifth lumbar vertebræ.

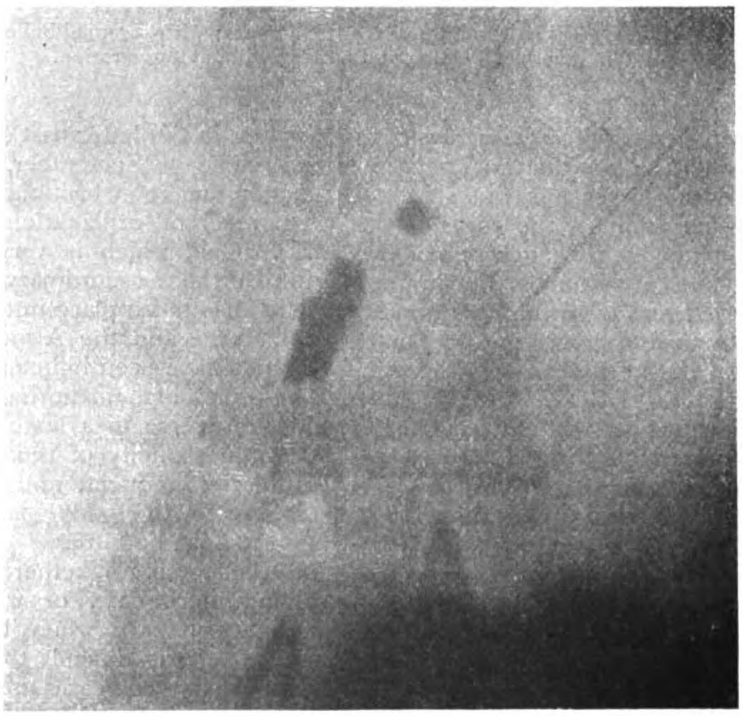

FIG. 14.—Shrapnel in spinal theca. The fragments were removed from among the cords of the cauda equina.

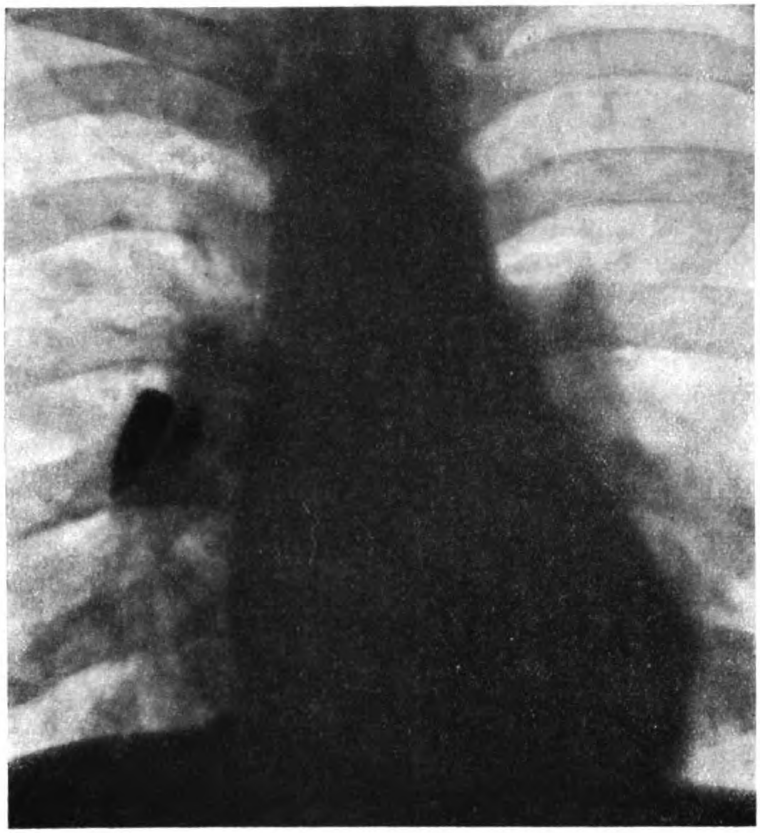

FIG. 15.—Sharp-nosed bullet in the hilum of the right lung.

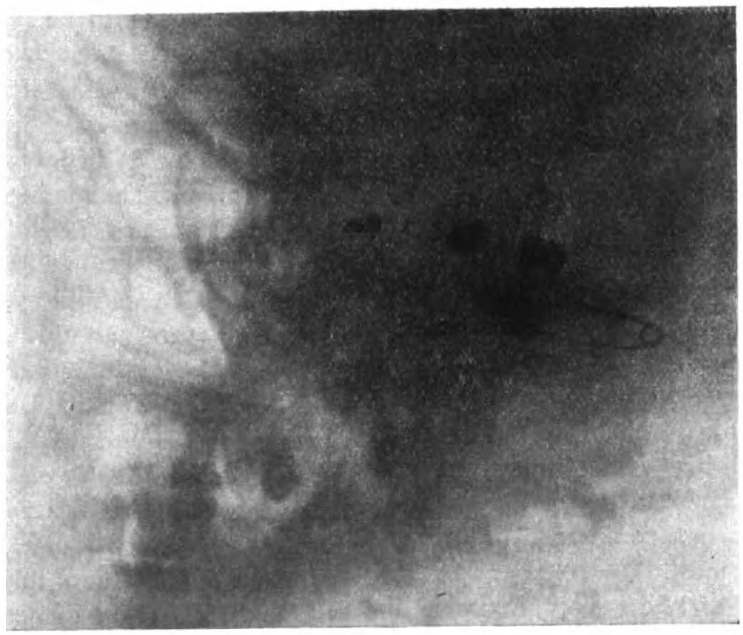

FIG. 16.—Portions of a ricochet bullet within the cranial cavity near the left sphenoidal fissure. They caused paralysis of the third, fourth, and first division of the fifth cranial nerves, but missed the sixth and seventh.

February 22nd, 1915.

"WHAT IS BRIGHT'S DISEASE?"*

By F. LANGRIDGE, M.R.C.S.

MR. PRESIDENT AND GENTLEMEN,—A short time ago I had a case of so-called "Bright's disease" in a patient with only one kidney, the other having been removed many years previously for calculus disease, and this caused me to look up the subject of Bright's disease. I was impressed, among other things, with the very vague and indefinite manner in which this term is used.

On looking up "Bright's disease" in the 'Encyclopædia Medica' I read, "See Nephritis," and on turning to 'Quain's Dictionary of Medicine,' under the heading "Nephritis," I find myself referred to Bright's disease; so that evidently these two authorities are not in strict accord as to the nosology of the disease.

In the article in the 'Encyclopædia Medica,' Dr. Nestor Tirard says: "Nephritis includes several forms of acute and chronic structural disease of the kidney, commonly associated with albuminuria and dropsy, with uræmic symptoms and other results of the interference with the normal work of the kidney."

He gives as varieties an acute form—*acute nephritis*, known also as acute desquamative nephritis, acute tubal nephritis, acute parenchymatous nephritis, and acute Bright's disease, most of these terms indicating that the chief brunt of the disease falls upon the parenchyma of the kidney—the glomeruli and the secretory or, to be physiologically correct, I suppose I ought to say, the *ex*cretory cells lining the urinary tubules. As a sub-variety "acute nephritis following scarlet fever" is also mentioned.

Secondly, a chronic form—*chronic nephritis*, known also as chronic parenchymatous nephritis, chronic tubal nephritis and chronic catarrhal nephritis, in which again, in the earlier stages, at any rate, the essential or secreting part of the organ is that which suffers most.

And lastly, *renal cirrhosis*, chronic interstitial nephritis, red granular kidney, or chronic gouty nephritis, in which the initial stage is supposed to consist of a hyperplasia of the connective tissue binding together the essential elements of the kidney, this new tissue by its subsequent contraction interfering with the function of, and ultimately bringing about a destruction of, the secreting portion of the kidney.

Thus under the term "nephritis," which he seems to regard as synonymous with Bright's disease, Dr. Tirard recognises three varieties—acute and chronic nephritis and renal cirrhosis.

Turning now to 'Quain's Dictionary of Medicine,' we find Sir John Rose Bradford advocating a more restricted use of the term "Bright's disease.' He defines it as "an acute or chronic inflammatory condition of the *cortex* of the kidney, often accompanied by secondary lesions in other organs of the body, especially the heart and blood-vessels," but he points out that all cases of nephritis are not to be regarded as cases of Bright's disease. As an example we may instance the so-called "surgical kidney," where an inflammatory condition of the kidney, proceeding even to suppuration, arises as the result of an ascending infective process originating in the lower urinary tract, caused, as a rule, by some obstruction to the flow of the urine. According to Rose Bradford, "the distinction between nephritis and acute Bright's disease depends essentially on the fact that the lesion in the latter tends to be progressive, and, further, that general effects such as dropsy occur, which are unknown in cases of simple nephritis." He acknowledges, however, that it is not always easy to draw such a distinction, for he says: "The difficulty of differentiating between nephritis on the one hand and Bright's disease on the other is so great that some writers consider Bright's disease and nephritis synonymous terms." He adds, however—and this is a very important point—"that one objection to the acceptance of this view (viz. that Bright's disease and nephritis should be regarded as interchangeable terms) is *that some forms of Bright's disease are of such a slow and insidious onset that they do not present an inflammatory character at any period of their course.*" (Italics mine.) But of this more later on.

He then goes on to describe an *acute form*, in which all the constituent elements of the kidney—the glomeruli, the epithelium of the tubules and the interstitial tissue—suffer, although to a different extent in different cases, sometimes one and sometimes another bearing the chief brunt of the

* Read at the Devon and Exeter Medico-Chirurgical Society.

morbid process; and a *chronic form*, of which he recognises two varieties, the so-called *large white kidney* and the *contracted* or *atrophic white kidney*, "sometimes called granular or cirrhotic," but he adds, "the latter terms are better restricted to the description of the true granular kidney, which is not here included under Bright's disease," and emphasises this point by saying "*the true granular kidney has no real relationship with Bright's disease and is an independent affection.*" (Italics mine.)

Hence we see that Dr. Tirard looks upon the cirrhotic kidney as a variety of nephritis, which term he apparently uses as synonymous with Bright's disease, while Sir John Rose Bradford regards it as an independent affection having "no real relationship with Bright's disease," and yet I make bold to state that the majority of the cases with which we meet in practice and which we label as Bright's disease belong to this particular variety.

There seems to be a natural tendency in mankind to collect and classify, evidenced in our youthful days in the direction of postage stamps, butterflies, and birds' eggs; but, unfortunately, Nature, who, in her evolutionary mood at any rate, rarely does anything *per saltum*, is sublimely indifferent to our artificial distinctions and refuses to be pigeonholed. Hence, although it is convenient and useful to divide these inflammatory conditions of the kidney into certain well-marked groups, these must be regarded not so much as a number of distinct and isolated pictures, but as a series of dissolving views, so that we get mixed forms, one type supervening upon and gradually replacing another.

But when we come to consider the first and the last terms of the series we have enumerated we cannot but be struck with the great contrast they present.

Look at the large kidney of acute nephritis, sometimes nearly twice the normal size—the enlargement due principally to an increase in the thickness of the cortex—the friable nature of its substance, the facility with which the capsule strips, and the smoothness of the underlying surface. Compare this with what we find in cases of interstitial nephritis, in which the kidney is so much *diminished* in size that the combined weight of both kidneys may be even less than that of a single normal kidney—the reduction in size again being the result of changes situated mainly in the cortex—the *firm* and *resistant* nature of the substance of the kidney, the more or less *adherent* capsule which, when torn off, exposes a *roughened* and *granular* surface, frequently presenting small cysts.

And this pathological difference is not more marked than is the clinical. Think of the sudden onset of a case of acute nephritis, the small quantity of urine passed, sometimes amounting even to complete suppression, its high specific gravity, the large quantity of albumen and casts present, the common occurrence of blood, and the nearly invariable accompaniment of a considerable amount of dropsy. Contrast this with what you find in cases of cirrhotic kidney—the *insidious* character of the onset, the disease having existed probably for many years before the case comes under observation; the *increased* quantity of urine of *low* specific gravity, containing little or *no* albumen and only occasional casts, with blood merely as an accident or complication; dropsy, too, to any extent is remarkable by its absence. The cardio-vascular changes, on the other hand, although present in the other forms, constitute such a prominent feature in cases of true granular kidney that it is often difficult when the patient first comes under observation to determine whether the case is one of primary heart or primary kidney, most, if not all, authorities regarding the condition of the kidneys in these cases as merely a local manifestation of a generalised arteriosclerosis.

As regards the small white kidney, this certainly presents a difficulty, for both pathologically and clinically it approximates to the true cirrhotic kidney, and although in some cases it is believed to be the terminal stage of the large white kidney, which in its turn may be, according to some authorities, the sequel of acute nephritis—both possibilities, as I will show later, most emphatically denied by Dr. Fred. J. Smith—Dr. Bradford considers that the small white kidney may exist as an independent affection, "many patients dying with a contracted white kidney who never suffered from dropsy, which is such a characteristic feature of the large white kidney."

And now as regards the independence of the large white kidney. Speaking of this Dr. Fred. J. Smith says: "It is a disease just as much *sui generis*, and absolutely independent of other kidney trouble as is, say, typhoid fever or cerebral glioma. . . . It never starts from or turns into any-

thing else, and may be called acute degeneration (fatty changes preponderating) of the kidney." ('Differential Diagnosis,' 1899, p. 218).

As I mentioned before, Dr. Bradford, presumably referring to the true granular kidney, says: "Some forms of Bright's disease are of such a slow and insidious onset that they do not present an inflammatory character at any period of their course." This naturally raises the question of what is meant by inflammation, and if we accept the definition that "inflammation is the reaction of the living tissues to an irritant, whether mechanical, thermal, chemical, or bacterial," the fact that the irritant in this particular case, though long continued, is of such slight intensity that at no time are the classical symptoms of inflammation—*rubor*, *calor*, *tumor* and *dolor*—present, hardly seems to justify us in declining to recognise the granular kidney as a variety of nephritis.

On the other hand, Professor Adami, writing in 'Quain's Dictionary of Medicine' on "Fibrosis," says: "With regard to the term *chronic interstitial inflammation*, while in the majority of cases we recognise that the undue development of fibrous tissue is brought about by the reaction to irritation, we cannot be certain that such inflammation is the one sole efficient cause in every case; there are cases in which we must assume some other cause." This, however, is essentially a question for the pathologist.

But, whichever view we hold, when we consider the marked differences, both pathological and clinical, between the desquamative and the cirrhotic types of nephritis, I do not see how we can fail to agree with Dr. Bradford when he refuses to classify them together under the one heading of Bright's disease.

You will notice that no mention has been made of the amyloid kidney, although some authorities regard this, also, as a variety of Bright's disease, but neither Dr. Tirard nor Dr. Bradford includes this under the heading of nephritis or Bright's disease, the latter remarking that "the amyloid kidney has only a superficial resemblance to Bright's disease and should be sharply separated from it."

To refer to other authorities would, I fear, only serve to make confusion worse confounded, for this seems to me to be a question of "*tot homines quot sententiæ*."

In the face of such conflicting statements and opinions, what is the unfortunate general practitioner and the still more unfortunate candidate for examination to do or say? I frankly confess I do not know.

The term "Bright's disease" is one of those unfortunate names which, although they help to perpetuate the memory of some celebrated physician, fail to convey to the student any idea as to the particular organ affected or the nature of the disease and, as I have shown, is differently interpreted by equally eminent physicians. One is naturally loath to part with these old names, especially when they have so often served to cloak for us what one might call a somewhat "blurred" diagnosis; but as Professor Huxley says, "There is no alleviation for the sufferings of mankind except veracity of thought and action." I therefore maintain that the time has arrived when, except, perhaps, for our patients and their friends, this term should be abandoned as inaccurate.

You will, naturally, ask me what I would suggest in its place. As a general practitioner I, of course, approach this subject from the clinical standpoint, and I would recognise an acute parenchymatous or tubal nephritis, about which all authorities seemed to be agreed, and two varieties of chronic nephritis, a parenchymatous or tubal form—the large white kidney found in "the large white patient" and an interstitial form, the small white kidney *and* the true granular kidney, for clinically it seems hardly possible to distinguish between these two; unless, as some authorities tell us, the small white kidney is a late stage of the large, when the previous history of the case would come to our assistance; but seeing that the treatment in both cases is practically the same, this is a distinction of but minor importance.

With regard to mixed forms which we are unable to relegate to any of these classes, I think the wisest plan is to follow the advise of Pudd'nhead Wilson, one of Mark Twain's celebrities, who says, "When in doubt, speak the truth."

Of course, if you care to include all these forms under the generic name of Bright's disease, I see no great harm in doing so; but what I specially plead for is a clear understanding as to the exact significance of this term.

March 1st, 1915.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE ON PUERPERAL ECLAMPSIA.*

By ROBERT JARDINE, M.D., F.R.F.P.&S.G.,
Professor of Midwifery, St. Mungo's College, Glasgow; Physician and Gynæcologist to the Glasgow Royal Maternity and Women's Hospital.

DURING the last two months, while you have been attending the bi-weekly clinics in the hospital, I have had under my care the following cases of eclampsia. Many of the cases have been seen by you, and in some you have seen the treatment being applied, but, as you are not here daily, you have not been able to follow up the cases to their termination. To-day I purpose to give you a very short history of each case, and in that way you will learn the results of our treatment. After all, the ultimate result of a method of treatment or an operation is the important thing, not only for the patient, but for the medical attendant. In your clinical studies, I would strongly advise you to try and follow up your cases to learn the ultimate results.

To obviate repetition in connection with the cases, I shall first state the ordinary routine of treatment which was adopted; and also state in connection with each case any deviation from that routine.

In the first place, the patients, on admission, require to be thoroughly cleansed, and many of them require this badly. When they are cleansed and brought to the labour ward, the active treatment is begun. The first procedure is to get the lower bowel cleared of its load by giving a copious enema. Then the stomach is washed out to clear it of any irritating contents, and a large dose of

* Based on a series of cases treated between November 7th, 1914, and January 2nd, 1915, in the Glasgow Royal Maternity and Women's Hospital.

mag. sulph. is left in it, and with this may be combined a large dose of chloral and bromides. Another method of giving chloral and bromides is by the rectum. In this series of cases these drugs were given by the stomach. The cleansing of the stomach will relieve it of its irritating contents, and the action of the mag. sulph. will clear the bowel from above, while the sedative action of the chloral and bromides will allay irritation in the nervous system. The urinary system is also at fault, and to deal with it we give saline transfusions of a couple of pints, usually under the breasts. The saline solution which we use contains one drachm each of sodium chloride and sodium acetate to the pint. This acts upon the kidneys and helps to flush the toxin out of the system, but we also make use of the skin to free the system of the toxin by causing sweating. Beneath the bed on which the patient lies there is a large copper pan which is filled with hot water. The patient is also surrounded with hot-water bottles. A hot pack will act in the same way, and in private cases it can be made use of. I must warn you that hot-water bottles must be carefully covered, and even then it is safer to have a blanket between them and the patient's skin. In eclamptic patients, the vitality of the tissues is so low that, if a hot-water bottle comes into direct contact with the skin, a burn may be caused. In applying poultices, this must also be borne in mind. Some years ago, I had a patient sent into hospital with the whole of her loins badly burnt from poultices having been applied over the kidneys. In an ordinary case the poultices would not have caused any damage to the tissues. In another case, one of my residents used chloride of ethyl to freeze the skin of the breast before inserting the transfusion needle. The result was, that wherever the ethyl touched the skin the parts sloughed, and it was many weeks before the patient could be dismissed. This was an act of mistaken kindness which I hope none of you will ever repeat. Sweating may be obtained by giving pilocarpine hypodermically, but the depressing action of this drug renders it dangerous, and it also causes a copious secretion of mucus in the bronchial tubes. More or less œdema of the lungs is always present, and an increase of mucus in the bronchial tubes is very serious, especially in a comatose patient. I have seen patients die of acute œdema of the lungs, so a drug which may cause this is to be avoided.

To control the fits chloroform is often administered, but it was not used in any of these cases for that purpose. The toxin affects the liver, and chloroform may intensify that effect, so it should only be given when an operation is necessary. By some morphia is used in large doses, but I only use it where there is great restlessness. I believe it tends to retard the activity of the kidneys and the bowels. I have seen a patient die of what apparently was morphia poisoning, although the doses given were small compared with what is sometimes recommended. Many years ago I tried veratrum viride, which, I think, was first introduced in America, but the preparations then in use were very unreliable, so I gave up its use; but when the active principle, veratrone, was introduced, I began using it again with extremely good results. It is most useful in cases with very high tension and pulse-rate. Within half an hour of its injection the tension of the pulse is markedly reduced, and the rate generally falls by forty or fifty beats. After its use the fits generally cease. As you will see, it was used in several of the worst of the cases in the series. The full dose is 1 c.c. hypodermically, but in some cases, especially in post-partum eclampsia, the effect may be alarming. At my request, Messrs. Parke, Davis & Co. have put it up in ½ c.c. doses, and I now use it in those doses, repeating the dose in a few hours if necessary. If alarming depression is caused, a hypodermic of strychnine will counteract this. I have used this drug in a large number of cases, and I have no hesitation in strongly recommending its use, especially in cases with a high tension and quick pulse. Bleeding from the arm I have frequently made use of in cases of high tension, but since I began using veratrone I have given up bleeding. As I have already stated, it lowered the pulse-rate by forty or fifty beats and also the tension, and, as you will notice, in most of the cases the fits ceased after its use.

In regard to the obstetrical treatment, we do not interfere, but leave nature to take its course. In the cases where labour has not commenced, no attempt is made to bring it on. In one case in the series delivery was effected by forceps, as the labour was prolonged. The child had died. In another case, in which there was unavoidable

hæmorrhage from placenta prævia, version was done because of the placenta prævia.

It is now pretty generally recognised that forcible delivery in cases of eclampsia does not give as good results as are got by leaving the delivery to nature, and only interfering if there is delay. Of course there are exceptional cases like the one with placenta prævia where interference is called for, but, broadly speaking, the less interference the better.

Last year I noticed a letter in the *Brit. Med. Journ.* from a general practitioner, saying that in an experience of twenty years he had treated some half dozen cases of eclampsia, and he strongly advised forcible delivery after the first fit. To me this seems a very big order. I have been treating cases of eclampsia for more than twenty years and have had to deal with several hundreds of cases, and my experience has taught me that forcible delivery is a mistake, except in a very few exceptional cases. You will notice that in a number of the cases in the series the fits began after delivery, so that the emptying of the uterus does not prevent or cure the fits. In one of the cases the fits began thirty-six hours after delivery, and in another on the third day, and I have seen cases in which they have begun as late as the fifth day of the puerperium. Judging from such cases, it seems a mistake to hurriedly empty the uterus, and experience bears this out. The present series of cases shows this as do many others with which I have had to deal. I have no desire to seem to lay down the law to you in this matter, but I give you the result of the experience of the treatment of hundreds of cases in which I have tried all the methods of treatment, and I have no hesitation in saying that forcible delivery in all cases is a mistake.

I shall now give you a very short history of the cases.

Case 1.—Mrs. C—, æt. 24 years, I-para, was admitted on November 7th in a comatose condition. She had been delivered in her home of a live child, but five hours after delivery she had had a fit. Her urine contained a large amount of albumen. She had one fit after admission. Her recovery was normal.

Case 2.—Mrs. C—, æt. 39 years, XI-gravida, was admitted on November 9th, after she had had four fits. Labour was commencing. A fit occurred as the patient was brought into the admission room, and two shortly afterwards while she was being prepared for treatment. In addition to the ordinary treatment, she was given veratrone. No more fits occurred, and the labour ended by natural efforts a few hours later. The child was premature, but alive, and it survived. In this case, it was noticed that the pulse-rate did not fall after the veratrone was given, but the patient vomited several times, and that may have kept up the rate. The albumen quickly disappeared from the urine, and patient made a good recovery.

Case 3.—Mrs. F—, æt. 17 years, I-gravida, was admitted on November 23rd, thirty-six hours after delivery of a live child. She had had fourteen post-partum fits and was deeply unconscious. During the time she was being cleansed and prepared for treatment she had ten fits, in fact, they were almost continuous. In addition to the ordinary treatment, she was given veratrone, and in half an hour the pulse rate was reduced from 120 to 80. Only one fit occurred after the treatment was begun. The albumen quickly disappeared from the urine, and the patient made a good recovery. You will notice that there were twenty-five fits in all, but only one occurred after the treatment was begun.

Case 4.—Mrs. McG—, æt. 30 years, I-para, was admitted on November 23rd, after delivery. The fits had begun on the third day after delivery, but we could not ascertain how many she had had. The bladder contained 70 oz. of urine, which had a good deal of albumen in it. The ordinary treatment was adopted, but she was not given veratrone. No more fits occurred, and the recovery was uninterrupted.

It is unusual for the fits to begin so late in the puerperium, but I have seen cases in which they have begun even at a later date. This patient was evidently suffering from retention of urine, and it is quite possible that the irritation from the bladder was the determining cause of the fits.

Case 5.—Mrs. D—, æt. 26, I-gravida, was admitted on December 3rd, after she had had three fits. She was seven months pregnant. The os admitted one finger. In addition to the ordinary treatment she was given veratrone. No more fits occurred, and the labour ended in the birth of a $3\frac{1}{2}$ lb. dead child on the following day. The albumen quickly disappeared from the urine, and the recovery was good.

Case 6.—Mrs. M—, æt. 32, I-gravida, was admitted on December 4th, after she had had two fits. She was deeply comatose, and we were informed that albumen had been present in her urine since May. The os admitted one finger. No veratrone was given. The fits ceased, but next day, when I visited the hospital, the patient could not see me at her bedside; she was quite blind, but next morning she had recovered her sight. Six days later the labour came on, and a live mature child was born by natural efforts. The patient made a good recovery, but a trace of albumen remained in the urine. It is probable that she was suffering from chronic Bright's disease. I have seen a good many instances of blindness in cases of eclampsia, but in all the others it has been during the labour, and the patients have been able to see when the coma passed off after delivery. The blindness is evidently functional, and not due to central lesion. The recovery of the sight within so short a time proves this, but in all the cases which I have had investigated by ophthalmic surgeons the report has been that there was distinct evidence of albuminuric retinitis, and that there would be a certain amount of impairment of vision. This is probably true of all bad cases of eclampsia, but I do not think that the cases which suffer from temporary blindness are more likely to suffer from impairment of vision than others. That is my experience as an obstetrician, but I am not an ophthalmic surgeon, and it is for them to decide this point.

Case 7.—Mrs. F—, æt. 26 years, II-gravida, admitted December 11th after she had had five fits. She was comatose and cyanosed, and her urine was loaded with albumen. No fits occurred after admission, and four days later a macerated premature fœtus was born by natural efforts. It weighed 4½ lb. The recovery was good.

In this case, I would like to point out to you that the urine contained a large amount of albumen. A good many years ago, it was observed, first, I think, by a German, that on the death of the fœtus the albumen disappeared from the urine. This may be true in Germany, but I have seen many cases of eclampsia with the urine loaded with albumen, some of them solid, in which the fœtus has been macerated.

Case 8.—Mrs. M. L—, æt. 24 years, I-para, was admitted on December 15th after delivery. She had had two fits. The first one had occurred an hour after delivery. She had two more within an hour of admission, and they then ceased. There was abundant albumen in the urine. She made a good recovery.

Case 9.—A. R—, æt. 28 years, I-gravida, was admitted on December 19th in labour, and delivered two and a half hours later by natural efforts, of a live mature child. The patient had not complained of any headache, and to all appearances everything was normal, but five and half hours after delivery she had a fit. Another fit occurred while the saline transfusion was being done, and two more within the next two and a half hours, when they ceased. She was not unconscious between the fits. There was a good deal of albumen in her urine. It quickly disappeared, and she made a good recovery.

Case 10.—Mrs. C—, æt. 36 years, VIII-gravida, was admitted on December 21st, seven months pregnant, and delivered next day. She had had sixteen fits before admission, and she was semi-conscious. The os was nearly half dilated. No more fits occurred, and next day a premature child of 3½ lb. in weight was born by natural efforts; it lived for three days. On the day after delivery the patient became maniacal and had delusions. One of her delusions was complimentary to the hospital, as she imagined that she was in Heaven, but as an offset to this compliment, she also imagined that her children had been murdered, and that their dead bodies were concealed in a corner of the labour room. She was given $\frac{1}{100}$ gr. of hysocine, which gave her some sleep, but when she awoke she was no better, so we had her transferred to Duke Street Hospital on the 24th, for mental treatment. There was a good deal of albumen in her urine on admission, but it had practically disappeared by the time she was transferred. We learned that her sister had died of eclampsia in May.

Insanity is a sequel of eclampsia, but the percentage of occurrence is not a very high one. Most of the cases I have seen have recovered.

Dr. Ivy Mackenzie has kindly sent me a detailed account of the treatment of this case, which I add. As you will see, she made a complete recovery.

Condition on admission.—This patient, admitted at 6.15 p.m., was in a very excited and resistive condition. Her talk was quite incoherent, as a

result of a confused maniacal state. She was very violent in her conduct, and seemed to be quite unconscious of the nature of her surroundings. Her physical condition was poor. She was pale and emaciated. Her heart and lungs were normal. There was a very faint haze of albumen in the urine. There was no discharge. Her mouth was in a filthy condition.

December 25th.—She was given a hypodermic of $\frac{1}{100}$ hyoscine, $\frac{1}{100}$ digitalin, $\frac{1}{8}$ morphia, $\frac{1}{100}$ atropine, and slept for several hours. She woke in a restless condition, and the hypodermic was repeated at 3 a.m., but she has slept very little since.

December 26th.—Yesterday she was very noisy and restless all day, and at 6 p.m. she was given another hypodermic, and had rather a better night. Was still restless and talkative. Passed sufficient urine. Bowels moved.

December 27th.—Patient yesterday seemed much better, was quieter, and showed some sensible appreciation of her surroundings. She slept very well from 8.30 p.m. till 4 a.m. this morning without sedative. She has taken her nourishment well.

December 28th.—Yesterday she was talkative and confused at times. She slept at night till 4 a.m., when she awoke in a restless and confused condition, and was given 20 gr. chloral and 30 gr. bromide. This morning she was quiet.

December 29th.—Patient is much quieter. She had the chloral and bromide mixture at 11 p.m. and slept well. This morning the confusion has disappeared considerably. She is taking her food well.

December 30th.—She is still talkative and somewhat confused, and a dose of the chloral and bromide has been given. Her excretions are normal, and she is feeding well.

January 4th.—Patient's condition has continued to improve, although interrupted by periods when she is talkative and confused. No sedative medicine has been given since the last note. She has been sleeping fairly well. She has a troublesome cough and is very anæmic, but she is taking her food well. To-day, for the first time, there is an appearance of depression as contrasted with the excitement which has marked her emotional state since admission.

January 7th.—Since the last note patient has continued to improve. She is taking her food well, her cough is better, and her colour presents a more normal appearance. Her emotional state is now quite normal.

January 9th.—Patient's mental condition is now normal. She is still somewhat pale, although during the last week she has improved greatly in this respect, and has put on flesh. She is very anxious to get home, and is to be dismissed to-day "recovered."

Case 11.—Mrs. K—, I-gravida, æt. 19 years, was admitted on December 27th in labour, in a comatose condition after she had had one fit. The os admitted one finger. Between 5 and 9.30 a.m. she had seven fits. In addition to the ordinary treatment, as the fits persisted, veratrone was given. There was another fit thirteen minutes after the injection of veratrone, and they then ceased. The urine was heavily loaded with albumen. Next day the patient was delivered of a dead child by forceps. She made a good recovery.

Case 12.—Mrs. B—, æt. 40 years, XIII-gravida, was admitted on December 29th after she had had three fits. She was seven months pregnant. The urine was full of blood and solid with albumen. She was comatose and very plethoric. A vaginal examination revealed that there was central placenta prævia. Up to this time there had been no bleeding. In addition to the usual treatment she was given veratrone. An hour later a fairly severe hæmorrhage occurred, so the vagina was firmly packed. Five hours later I removed the packing and found the os sufficiently dilated to do version. As the child was dead I perforated the after-coming head. The placenta was very adherent, and I had to strip it off the lower uterine segment. The patient made a good recovery.

This is the first time I have ever seen placenta prævia in a case of eclampsia. The combination must be an extremely rare one. Cases of placenta prævia are just as liable to albuminuria as ordinary cases, but very many of them have bleedings by the seventh month, and this will relieve them to a large extent of the risk of fits occurring. In addition, very few of them go on to full time, and this also lessens the risk of fits occurring.

Case 13.—Mrs. McD—, æt. 20 years, I-para, was admitted on January 2nd of this year, after delivery of a six months dead fœtus. She had had seven fits post-partum. A fit occurred while she was

being washed, another while an enema was being given, and a third when the stomach was washed out. The urine contained a moderate amount of albumen. No more fits occurred, and the patient has made a good recovery.

There are many interesting points one might discuss in connection with these cases, but we can only deal with a few. You will notice that nine of the patients were pregnant for the first time, and of the multiparæ, one was pregnant for the eighth time, one for the eleventh, and one for the thirteenth time. The condition is much more liable to occur in first than in subsequent pregnancies. It is also more frequent among single than among married patients. However, in this series, there was only one single woman. In five of the cases the fits began after delivery. This is a larger proportion than usual. By some it is held that in post-partum cases the prognosis is much graver than in others, but this has not been my experience. The number of fits varied from three to twenty-five. As a rule, if there are ten or more fits the prognosis is bad, but I have had a patient recover after she had had over 200 fits, while, on the other hand, I have seen a woman die in a first fit. The case was one of advanced phthisis, which I saw in consultation a week or so before the labour was due. The breathing was very much embarrassed. I found the woman sitting propped up in an armchair. Her pulse was good, but intermittent. There was no headache. As the uterus contained a considerable amount of liquor amnii I decided to rupture the membranes, and asked the nurse to get the patient into bed. When I had washed my hands the patient had just placed her head on the pillow, and she at once took a typically eclamptic seizure. In the tonic stage her breathing ceased, and I at once began artificial respiration. The arms were rigid when I pulled them above the head. She never breathed again. In another case I was called to, I found the patient dead. Her husband, a medical man, told me that he and his wife had had dinner together, and then his wife had said that she was not feeling quite well and that she would go to bed. She did so, and died in a fit a few minutes later. She was nearing her confinement.

In regard to the urine, the important point to bear in mind is not the amount of albumen present, but the amount of urine which is being excreted. If the kidneys are acting well the chances for the patient are good, no matter how abundant the albumen may be. In Case 12, the urine was full of blood and solid with albumen, and yet the patient made a good recovery.

In addition to these thirteen cases, we have had one other case of fits: I believe they were epileptic. There was no albumen in the patient's urine. I have never yet seen a case of true eclampsia in which there was no albumen in the urine. We also had a number of cases of albuminuria, and two of them had the premonitory indications of eclampsia, such as headache and twitchings, but they were treated and delivered safely without fits occurring. As you know, Prof. Cameron and I admit patients on alternate days. During the two months he admitted eight cases, making twenty-one in all. During that time 266 patients were admitted to the Hospital. That is an enormous proportion of cases of eclampsia. You can see how misleading our hospital statistics would be in regard to the frequency of eclampsia. In private work one case in 500 will be nearer the mark than one in 13.

There is just one other point I shall call your attention to. Any of you who are keen observers will have noticed, that on some of the days of your visits there were several cases of eclampsia in the labour room, and these days were specially cold ones. Whenever there is a sudden lowering of the temperature we expect cases of eclampsia to be sent in, and they generally turn up all right. The same thing occurs in summer weather. I have watched this for years, and many times I have told my residents that eclamptic cases would turn up within a few hours, and the forecast has generally been correct. These patients have albuminuria, but the kidneys and skin are still excreting sufficiently freely to relieve the system of the toxin. Then there is a sudden fall in the temperature, the skin gets chilled, and a greater strain is thrown upon the kidneys than they can bear, and the result is an increase in the toxin and an explosion in the form of fits. In connection with this there are two practical hints I wish to give you. The first is, in treating cases of albuminuria of pregnancy, see that your patients are warmly clad, and that they are not exposed to draughts; and the other is, if you are sending an eclamptic patient into hospital, see that she is well wrapped up, and has plenty of protected hot-water

bottles about her. We find that patients brought from a distance in the ambulance have a poorer chance of recovery than those which are in the hospital at the time of seizure. I can recall two cases which were brought into hospital after they had lain on the floor at home for several hours. In both cases the women were alone in their houses, and they had been seized when they were out of bed with only their night-dresses on. They both died.

Note.—On the afternoon of the day on which this lecture was given two more cases were admitted, and since then there have been three additional ones. Prof. Cameron has also admitted two more, making twenty-eight cases in three months.

March 8th, 1915.

Hæmorrhagic Symptoms in Typhoid Fever.—Braillon and Bax state that apart from the epistaxis which may occur at its onset and intestinal hæmorrhage due to the specific ulceration of the bowel, typhoid fever may assume a true hæmorrhagic aspect which dominates its clinical course. This hæmorrhagic condition may assume two distinct types according as it appears early or late. These differ also in character. The early form is the more severe, in fact a malignant hæmorrhagic type, similar to that which occurs in other infections. The later occurs during defervescence or convalescence in cases which have given no indication of unusual severity. It usually lasts about six or eight days, ending most frequently in recovery. The following is an unusually severe case of this type, which even proved refractory to transfusion of blood.

A girl, æt. 16 years, was admitted to hospital on October 28th, suffering from typhoid fever, which had already lasted fifteen days. Her illness began with scarlatiniform erythema over both legs, succeeded by vomiting, diarrhœa, and insomnia, but there was no epistaxis.

On admission the temperature ranged about 104° F., the pulse about 80. The erythema had disappeared, the urine was clear and abundant, but rose spots were present on the abdomen and Widal's reaction was positive at 1 in 50. She was treated by milk diet without baths, and the only drugs administered were urotropin and adrenalin, with a few doses of pyramidon. On November 7th the temperature began to fall and convalescence seemed imminent.

On November 11th the urine was slightly red, which was ascribed to the pyramidon, although none had been given for some days. On November 13th there was slight bleeding from the gums, attributed to the use of the tooth-brush, but the urine was more highly coloured. In the evening epistaxis occurred, and the bleeding from the mouth and gums continued. A mixture containing chloride of calcium and ergotine was prescribed. On November 14th, considerable intestinal hæmorrhage occurred, and the oozing from the gums continued. The urine contained much blood. The temperature was normal, but the pulse was 120 and very small, and the skin and mucous membranes were very pale. She was given caffeine and a hypodermic injection of serum with ·04 grm. of emetine hydrochloride. On November 15th the bleeding from the nose and mouth continued, as did the hæmaturia, but there was no further hæmorrhage from the bowel. The patient was in a state of torpor and appeared almost exsanguine. The pulse varied from 130 to 140 and was almost imperceptible, while the temperature had fallen below 98·6° F. An injection of 500 c.c. of saline solution was administered subcutaneously. In the afternoon she was in a precarious condition, and the continuance of the hæmorrhages suggested the advisability of transfusion of blood. This was performed by connecting the median basilic vein of the patient with the radial artery of her father by means of the cannula of Elsberg. The connection was maintained for forty-five minutes, by which time the donor had become pale and rather faint. The patient felt better and showed slight colour in the cheeks, while her pulse fell to 100, but was still very small. Next day she showed marked improvement, the skin and mucous membranes being well coloured, the pulse 90, well filled, and the temperature 101° F. The bleeding from the nose and gums had ceased, and the hæmaturia was less marked than before the transfusion. The bleeding from the gums recurred during the night, and on the following day the hæmaturia again increased. In the afternoon she vomited a large clot of dark blood. An injection of ·04 grm. of emetine hydrochloride was again administered. On Novem-

ber 18th hæmatemesis recurred and the oozing from the gums continued in spite of the application of perchloride of iron. The hæmaturia was also very abundant. The forearm at the site of transfusion had become œdematous, and on exposing the wound a soft clot protruded. In the afternoon profuse bleeding occurred from the wound and could not be entirely arrested. Next day her condition appeared to be hopeless. The oozing from the wound and from the gums continued, as well as marked hæmaturia. The pulse was 160 and almost imperceptible. A slow instillation of 500 c.c. of saline solution containing 47 parts of glucose in 1000 was given by the rectum. This was replaced in the afternoon by a solution containing 1 per cent. of gelatine, of which a litre was given during the afternoon and evening with perfect tolerance. A hypodermic injection of 10 c.c. of electrargol and another dose of emetine were also given. On the following day the temperature was 100° F. and the pulse 120. The hæmorrhages had ceased suddenly and simultaneously during the night. The gums were almost normal and the urine was clear and free from blood by chemical and microscopical examination. Another 500 c.c. of glucose solution was given by the rectum. Next day the improvement was maintained and no further hæmorrhage occurred. She gradually regained strength and left hospital on January 24th in a perfectly satisfactory state of health.

On November 15th, before transfusion, the red corpuscles numbered 2,700,000. The puncture in the finger from which blood was drawn bled profusely and the oozing had to be arrested by pressure. On November 20th, the day after the cessation of the hæmorrhages, dry blood films showed the following percentages: Neutrophile polynuclears, 79; large mononuclears, 7; lymphocytes, 10; eosinophile myelocytes, 1; eosinophile polynuclears, 1; nucleated hæmocytes, 2. This observation was completed by an enumeration of the corpuscles two days later, when the red cells were 1,200,000 and the leucocytes 16,000. Four days later, the number of red corpuscles had risen to 2,800,000. The day before she left hospital, January 23rd, the red corpuscles numbered 3,080,000 and the leucocytes 10,400, while the hæmoglobin was 75 to 89 per cent. The differential leucocyte count was as follows: Neutrophile polynuclears, 64; mononuclears 22; lymphocytes, 14; while eosinophile polynuclears and mast cells were absent.

The writers point out the unusual severity of this rare condition and the failure of the transfusion of a large quantity of blood to arrest it in spite of the great temporary improvement which occurred. The striking arrest of the hæmorrhages by the instillation of gelatinised saline solution *per rectum* shows the value of this method even in apparently hopeless cases.—*Bull. et Mem. de la Soc. Méd. des Hopitaux*, p. 194.

Diseases of the Heart. By John Cowan, D.Sc., M.D., F.R.F.P.S., Professor of Medicine, Anderson's College Medical School, Glasgow. London: Edward Arnold. 15*s.* net.

The diagnosis of heart disease has now been reduced to an exact science. In this book, in addition to the author's work, Dr. W. T. Ritchie, F.R.C.P.Edin., is responsible for the chapters on "The Electrocardiograph" and Dr. Arthur J. Ballantyne, M.D., Glasgow, for the "Ocular Manifestations in Arteriosclerosis." The myocardium as a separate and distinct study is rapidly inheriting its own territory, and the somewhat vague terms of "tired," "irritable," "over-strain," etc., must soon be displaced by definite entities. The subjective symptoms of heart mischief have not till lately received full recognition.

From 1794 to about the present date, or approximately 120 years, the name "Cowan" has connoted the Royal Infirmary of Glasgow, from which, in the work before us, this institution has supplied excellent material for a full and monumental study of an increasingly interesting subject. It is high time the use of the ophthalmoscope became more general, and part of the regular equipment of the medical student. For Dr. Cowan's book we have nothing but praise. The subject of "Angina" might with profit be amplified in a future edition. The book is written from the practitioners' standpoint. It is light to hold, easy to read, abundantly illustrated, and will amply repay close study.

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We regret that owing to the war the number of pages must be diminished for a time.

INJURIES OF THE SEMILUNAR CARTILAGES.*

By C. H. FAGGE, M.S., F.R.C.S.

GENTLEMEN,—The subject of injuries to the semilunar cartilages is, in England, an old one. Every description of this lesion in the text-books begins with the statement that more than a hundred years ago Hey put together the clinical picture of a traumatic lesion of the knee, which he called internal derangement of the knee-joint. There is no doubt that, in early cases, traumatic internal derangement sufficiently defines the mental attitude of the surgeon; the term is now gradually dropping out but seems to have something to commend it, because in the early stages diagnosis is almost impossible, as is shown by the statement of that well-known orthopædic surgeon, Robert Jones, of Liverpool, who wrote that he never opened a knee-joint with any sense of security or with a certainty that he would find the lesion which he expected; also that he was never disappointed to find that the condition which he had diagnosed before operation was not present. When a surgeon with the reputation and clinical experience of Robert Jones makes such a statement, it must have considerable weight, for in the same paper he said he was writing an account of his experience of five hundred operations of the knee.

About a year ago I was interested in the question of injuries of the knee, and read most of the literature which was available in London on the subject of such injuries. It struck me as very curious—and perhaps fortunate for me—that practically all the literature on the question was in English. A well-known French text-book

* Clinical lecture delivered at Guy's Hospital.

dismisses the subject in less than a page, describing it as an injury to Anglo-Saxons given to violent exercises, particularly football. Many of you know the very comprehensive literature which, from time to time, is put out by the Mayo clinic. Four large volumes have been issued since the year 1909, but there is no single word upon any injury to the knee-joint in these, and the curious exception to the fact that this is an injury of the knee-joints of those given to violent games is, that it does not seem to take place with any frequency in America.

During the week of the Clinical Congress last July, I happened, partly on purpose, but more by chance, to operate on two supposed lesions of the semilunar cartilages. And the reason I did it was because in my short visit to the States I never saw an operation on the semilunar cartilages, or upon any traumatic condition of the knee-joint; therefore, I thought it might be of interest to members of the Congress. One surgeon from New York, whom it is my pleasure to count as a friend, said that in his clinical experience these operations were very infrequent.

There was practically no German literature on the question, but one of the best known text-books in German (Koenig) says that their knowledge of the condition was largely due to Mayo Robson. And it is one of many subjects which Mayo Robson's vast clinical experience and considerable operative technique did much to illuminate; he labelled the condition from what he saw in the North as "miner's knee." It was not until about the year 1900 that the knee-joint was explored with any sense of security. About that time Sir Arbuthnot Lane wrote a paper on injured semilunars, and suggested that they offered a focus for the onset of knee-joint tuberculosis, a view which has recently been supported by Robert Jones. In the 'Proceedings of the Royal Society of Medicine' for 1912, there are two interesting papers on injuries to the semilunars; one came from Mr. Martin, of Newcastle, where they know more about injuries to the knee than in any other town in England. The report of the Royal Victoria Infirmary, Newcastle-on-Tyne, for the year 1911, contains details of 156 cases which were operated upon for injuries of the semilunar cartilages. It is interesting that only two of them were women; in 151 instances the internal semilunar was injured, and there were six torn external semilunars. In one case both the internal and external semilunars were operated upon.

Martin's paper above mentioned deals with his own experience of 449 cases; of these 62 per cent. were miners, and 18 per cent. were due to football accidents. And then comes the most important fact, to my mind, connected with injuries of semilunars which I have learned during the last two or three years, namely, that in 95·5 per cent. the cartilage was torn. To talk of a displaced or detached semilunar cartilage is, in itself, a misnomer, and it is a much worse mistake to say so than appears at first sight. Because if the substance of the cartilage is torn, and in every case a portion of cartilage remains in contact with the peripheral capsule, it is pretty clear that any effort—and the efforts were very frequent in the early surgery of the semilunar cartilage—any effort to suture it back in position must more or less necessarily fail, because union of cartilaginous tissue after tearing is likely to be bad; cartilage is a tissue which does not heal well. The way in which we talk about detached semilunar cartilages is only one instance of a very common feature of surgery. We are inclined to operate for a condition, to say what we think there is to remove, and to say what we expect to find wrong. We do not often—and the older we get the less inclined we are to do this—modify our preconceived opinion by the conditions we find. And one of the most important mechanical factors on which these mistakes depend is, that so many people think they can find out exactly what is wrong with the knee-joint, however minute the incision. If you see surgeons like Jones, Martin, and Rutherford Morison operate upon a semilunar cartilage, you will appreciate that they only do so through a very free incision.

The discussion at the Royal Society of Medicine was interesting because Mr. Walton, of the London Hospital, put forth the idea that the commonly accepted view as to the way in which the semilunar cartilage was injured was wrong. He argued that the most important feature in an injury of the semilunar cartilage—and when I say semilunar cartilage I nearly always mean the internal one—was rotation. And being interested at the time mainly in mechanical studies, he perhaps pushed his anatomical inferences a little too far. He argued that rotation in the knee-joint commonly

takes place only at the end of extension, or at the beginning of flexion, just as you may have read in Cunningham's 'Anatomy,' where a very good account of the movements of the knee is given. On this premise Walton argued that injuries to the semilunar cartilage only take place with the knee hyper-extended.

It is rather sad that we are always inclined to think that because a young man says a thing which is not in accordance with our pre-conceived ideas, that it is necessarily wrong. I do not say that Mr. Walton is right in this matter, but I think his critics might have realised that their own pre-conceived conception as to the method by which these injuries were produced was not logical nor conclusive in all its aspects, and that there might be a good deal of truth in what Mr. Walton said.

There is, however, no getting away from the fact which Mr. Martin, of Newcastle, well brought out, that the injury as commonly seen in the North occurs with the knee flexed, because in that part of the world the coal seams are so low that all the men work practically in the sitting posture. I gathered from an old Newcastle resident, who had seen a good deal of surgery there as Mr. Morison's assistant, that the miners work practically sitting on the right heel, and that in most cases a fall of coal actually pushes the knee inwards.

There is no doubt that, as we see it down South, the injury to the semilunar is due to indirect violence occurring with the knee semi-flexed, abducted, and generally with the femur rotated inwards. But I think Mr. Walton has done the subject a service by pointing out that at the time the person is injuring his knee in this way, he is making an effort to extend it. Imagine a tennis player or football player momentarily poised on one leg, with the knee slightly flexed and abducted, when someone tries to collar him, or hits the ball on the other side of him, and in either case he tries to turn, forcibly rotating his knee. But while he is trying to turn, it is clear he must be making an effort to extend the joint.

When the knee is flexed, the capacity of the joint is greatest; thus, all inflamed knee-joints adopt the position of semi-flexion, and when the capacity of the joint is greatest, there is the greater need for the packing structures to fill up the different spaces, and the semilunars, as one of these packing materials, are drawn towards the centre of the joint. As the knee is extended these cartilages, particularly the internal, are drawn out towards the periphery by the stretching of the lateral ligaments. Let us suppose, then, that in the semi-flexed position the central concave border of one cartilage is by chance nipped between the two bones and the sudden effort at extension drags the lateral ligament and the circumferential convex margin of the semilunar towards the surface; in the case of the internal it will be inwards, and in the case of the external it will be outwards. And so it is split, as you would tear a piece of linen, by two forces acting in opposed directions. It is important to realise that in nearly every case there is a tear. I have no doubt that rotation is a very important feature in its production, and probably one of the factors which influence the percentage proportion of the injuries to the internal, as compared with the external semilunar is the fact that rotation mostly takes place on the inner side of the knee-joint. You may remember the account of the internal condyle of the femur screwing its way home, as described in Cunningham's 'Anatomy,' which will help to account for the figures from Newcastle, 148 internal semilunars injured as against six external, which shows the great proportion of internal as compared with external semilunar injuries. The most important anatomical fact, however, which accounts for this disproportion is, no doubt, the firm attachment, circumferentially, of the internal semilunar to the internal lateral ligament. This is not found on the outer side, for the external lateral ligament is kept away from the external semilunar by the popliteus tendon and the synovial sac which intervenes. You can see in most anatomical preparations of the knee-joint that a shrinking inwards towards the centre of the joint does take place in flexion, and that the cartilages tend to be expressed out of the joint cavity in extension. You can almost demonstrate that for yourselves by flexing and extending your own knee.

There seems to be no real reason why the right should be more often affected than the left, but so it is. Leaving now anatomical considerations, of which I have only touched the fringe—because movement of the knee-joint is a very interesting question—we come to a consideration of the symptoms which are produced by injury of the semilunars. Rutherford Morison (CLINICAL JOURNAL,

April 9th, 1913) gave a very clear symptom-complex, which he regarded as typical of semilunar injuries, as follows: (1) A really severe injury which is associated with considerable pain followed by effusion into the joint, and which lays the individual up for a period, perhaps for a fortnight in bed, and will incapacitate him for several weeks from following his ordinary occupation. (2) This injury, so severe as to be well remembered, is followed by a period of complete freedom, only to be again interrupted by troubles in the knee, which are often due to slight causes, are of short duration, and sometimes associated with effusion, and which come on quite unexpectedly.

I happened, by chance, to operate on a girl this morning who was regarded by her doctor as having an injury to the internal semilunar. She is, in some respects, an exception to what I have been telling you, because she is not only a girl, but a French girl, but her history is in most respects typical of what Mr. Morison says, and will serve to illustrate his points. I saw her a fortnight ago when she told me that in the early spring of 1911 she was playing tennis in England; when jumping over the tennis-net she caught her foot in the top of the net, which accident, in spite of almost immediate treatment, was followed by effusion, and she thinks she was in bed three weeks, and was not allowed to do anything for five weeks. In the next year she slipped on the stairs and hurt her knee again. A third attack occurred in 1913, and in May of this year an interesting thing happened, because, owing to some trivial cause which I have forgotten, her knee became locked. A fortnight ago she was kneeling in church, and when trying to rise from that posture her knee locked again. She sent for the doctor, who unlocked it. When I saw her next day the knee-joint was full of fluid. Her mother said it was certain that after any excessive exercise this knee-joint was larger than its fellow. The knee-joint was opened, the internal semilunar found torn anteriorly, and the anterior part removed.

Rutherford Morison, in his account of these cases, does not lay stress on one point which is prominently given in some books as pathognomonic of semilunar injury—that of locking; and if you look at the question logically, one would expect Mr. Morison to be right. Any loose body in the knee-joint which happens to come between the opposed articular cartilaginous surfaces may produce locking, just as by putting a stone in one of the hinges of a swing-door you will prevent it shutting, or by putting a stone on the other side you will prevent it being opened. Locking means a limitation of movement in one direction. It is not unusual to get a history of locking with semilunar injuries, but locking in both directions is very rare; thus, this girl, who was a good example of locking towards extension, could allow her knee to be freely flexed beyond the right angle, but it could not be extended to the full extent.

The piece which is torn projects and gets in the way between the cartilaginous surfaces, and generally rather to the front of the joint, and so prevents extension.

There is only one variety of injury to semilunar cartilages which apparently does not give a typical history, and that is the variety which Morison called the "bucket-handle" fracture. Here there is a longitudinal tear right along the semilunar, and the free piece remains attached in front and behind, but is not connected with the rest of the cartilage at its peripheral margin. Early in the summer I was operating upon another girl sent in as suffering from a torn semilunar. Do not think from this that my experience of this injury in girls is a large one; I have only operated upon three girls for it, and I own that even this number in my small total experience of this injury is exceptional and shows that one cannot found any statistical evidence on a small number of cases. The history of this girl did not quite conform to what I have told you of semilunar injuries. It was that the condition was of a year's duration, and the attacks had been so frequent and the disability so continuous as to interfere with her occupation as a servant. I did not feel definite enough about the condition to justify me in refusing to operate, but, candidly, I was quite sceptical as to her having an injury of the internal semilunar; I thought she had one of the other conditions which may easily be confused with injury to the semilunar, and said so at the time. On opening the joint I found what I took to be a normal semilunar, but between the condyles of the femur there was a strip of cartilage passing from back to front.

This was displaced outwards into the intercondylar notch, and, in fact, looked like the handle of a bucket. I have had experience of

only two* of those cases, and therefore know very little about the condition from personal experience; but Rutherford Morison rather makes a point that in cases in which this type of lesion exists the intermittent, interrupted history characteristic of the common type is not obtainable.

With regard to the diagnosis of the condition, there is even now, as you will have gathered, some considerable doubt. In his more recent papers Mr. Robert Jones does not dilate so much on the difficulty of diagnosis or on the uncertainty with which he now opens a knee-joint, but I do feel very strongly that one should realise that there are other conditions which are easily mistaken for injury of the semilunar.

The most common of these is a form of chronic synovitis, in which all the synovial fringes—the ligamentum mucosum, for instance—are hypertrophied and thickened. One of these fringes will often produce disability by being picked up between the two articular surfaces, and will give a history which is easily confused with that of a case of injured semilunar. But if you dissect the history carefully you will usually find that there has not been one severe well-remembered injury which has been followed by a succession of slight attacks. This condition of villous synovitis is often part of a general traumatic arthritis of the knee, which is particularly common in middle-aged men, particularly working men at this period of life who have been out of work for some time and then go to a hard job for which they are not physically fit. They are very apt to get a progressive incapacitating arthritis of the knee, which, however, they are very apt to attribute to injury, and sometimes the condition is mistaken for an injured semilunar. In progressive traumatic arthritis of the knee operation does harm, for I know of two or three cases that have been operated upon and in which the disability has been increased by the operation. If a man over 30 comes under my care with a knee injury, I am doubtful about it being a torn semilunar. Of the cases on which Mr. Walton's paper was formed, fifty-three were below 30, and twenty were over 30 years of age. I should not have thought there was anything like this approach to equality of numbers, but rather that the proportion was ten under to one over 30.

Less common traumatisms of the knee-joint are pieces of cartilage knocked off, forming those fugitive loose bodies (joint-mice) in the knee which are so difficult to find when the knee-joint is opened. There is also the organisation of a synovial exudation into the knee, giving rise to those loose bodies known as "melon-seed bodies." Even isolated synovial fringes which have hypertrophied may give rise to difficulty in diagnosis, but are not important because in the majority of instances the treatment is the same. I remember a case illustrating this, that of a sailor, in which I opened the knee-joint with the idea that he had an injured internal semilunar. I found one of the infra-patellar pads of fat, which was very thick; it was, in fact, a hypertrophied ligamentum mucosum torn away from its attachment to the inter-condylar notch, and, the cartilages both being perfectly sound, this was removed. The man had no subsequent attacks during several months in which he was under observation.

Some time ago I was very much puzzled by a little girl, æt. 10 years, whose knee made an extraordinary noise when she bent it. I felt certain there was a loose body in the joint, which, in certain positions, got between the articular surfaces, and was released with a snapping noise. There is a condition of the hip which the French call "hanche à ressort," and so a similar condition of the knee may be called "genou à ressort." I find, as is nearly always the case, that someone else has described the same thing, and put it down to the right cause. In my case the correct cause was only made known to me by Mr. Townrow, who was then Surgical Registrar. When he saw the patient in the ward he said he thought he knew what the trouble was, as he had seen two or three like it, and in each case, the external semilunar was loose.

I found later that Mr. Robert Jones ('Annals of Surgery,' 1909), describes exactly the same condition as "the snapping or clicking knee, a condition very puzzling, . . . is a loud click on acute flexion and sometimes on complete extension." Jones goes on: "This is described as trigger-knee because of its characteristic snap. . . . The cause of the difficulty is generally ascribed to disturbance of movement of the external semilunar.' In my little girl it was only heard when the knee

* A third case of bucket-handle fracture, also in a servant girl, has since been in my wards. The history was short and continuous.

was nearly approaching full flexion; then the click was heard, and the whole knee-joint seemed to stagger; similarly soon after extension began, the sound recurred and then the leg came forwards noiselessly and freely.

The external semilunar was found to be at fault, for the posterior part, where it is normally free, was mobile and thickened. When the anterior part of the cartilage had been cut through, the free thickened part could be pulled forward so as to become impacted between the articular surface and in both extension and flexion the clicking noise could be caused by its release. This was removed, and the noise did not recur, either on the table or when she was sent out of the hospital.

Treatment.—During the initial attack of injury to the cartilage, its presence can be only suspected. There is a complete agreement amongst authorities that operation is not justified in a first attack; grave suspicion of semilunar injury may be aroused, but this does not justify operation. I have many times seen men in whom I have suspected or provisionally diagnosed torn semilunars, but who on later visits have shown a normal knee and a freedom from attacks for months or years. During the acute attack immediate treatment is very important. Much pain is avoided by the early application of firm elastic pressure over the knee by a large covering of cotton wool and a firm bandage. A rubber bandage if obtainable is best, and the joint is maintained as nearly extended as comfortable without a splint. The firm bandage may cause some swelling of the foot, but that does not matter if it causes no pain.

With regard to the method by which, if the knee is locked, you should reduce the locking, the great thing to do is to very firmly and fully flex, and then extend, rotating inwards a little. Most of the loose bodies which are locked between the joint surfaces can be so released.

I have very little to say about the non-operative treatment. Instruments have been designed of which the best is that known as Hawksley's splint, which is recommended in most text-books. These, no doubt, merely reflect the opinion of some years ago, and nobody would now advise the instrumental treatment of a torn semilunar cartilage, excepting under unusual circumstances, as in a patient who was old or debarred from any active exercise.

With regard to the operative treatment, there seems to be an equal division between those surgeons who make a transverse incision extending from the inner border of the ligamentum patellæ to the internal lateral ligament and those who make a vertical incision. In my thinking it does not matter which is employed so long as the incision is free, allowing the interior of the knee-joint to be thoroughly examined; this is best done with the joint in the fully flexed position.* A free vertical incision is my practice, extended from the upper limit of the tibia on the inner side of the patella well into the aponeurotic expansion of the vastus internus. By this means the interior can be seen and the position of the cartilage can be judged as the joint is flexed and extended. Whitelocke ('Sprains and other Injuries of Joints') and Jones remove only the loose part of cartilage; Killock in a personal communication advises removal of the whole meniscus, and for this object uses a curved oblique incision with the leg over the table-end. Many surgeons advise the application of a tourniquet to the thigh before operation. It always seems to me that if you suspect bleeding during an operation, it is best, as it is surgically sound, to see the bleeding spot and to deal with it. I would rather see where bleeding is, and deal with it at the time, than that there should be any risk of bleeding taking place into the joint.

Yet in spite of care in this respect the only complication to be feared is distension of the joint with blood; only a year or two ago a man in Guy's was operated upon by me for an injured semilunar cartilage, and this was followed by an effusion of blood into his knee. His temperature rose on the second and third day with pain and tenderness, and this was so alarming that I opened the knee-joint again. Much blood-clot, which fortunately proved sterile, was turned out and the joint was sewn up again. In my experience, such post-operative hæmorrhage is the only complication in these cases to be feared. It may usually be avoided by applying a large pad as dressing, and a plentiful layer of cotton-wool lightly secured by a Domette bandage. The vessel which is likely to bleed is one taking part in the anastomosis between the inferior external and the superior internal articular arteries behind the ligamentum

* Jones and others allow the leg to fall over the foot of table, the knee being flexed to a right angle. I have tried but do not favour this method.

patellæ; as the incision comes down on the inner side of the patella, this vessel is met with in the extra-synovial fat, not in the substance of the synovial membrane. It is common to find a fairly large vessel in this position, which is usually divided, and if after the operation is completed by the removal of the affected piece of cartilage, the capsule is sewn up in a separate layer from that suturing the synovial membrane, the vessel can usually be controlled by the second suture, and no ligature is necessary.

It is of great importance that one should never ignore the risk of sepsis in a joint, but at the present day I do not think there is any probability of infection of a knee-joint under ordinary conditions. The importance of this question, which many text-books discuss at length, has largely died out at the present time. As far as I know, every case of this lesion on which I have operated has been, during the time it has been watched, free from recurrence of attacks, and movements and strength of the joint have returned to the normal. Mr. D'Arcy Power has reported the after-histories of eighty-nine cases at St. Bartholomew's Hospital, and found that in seventy-three there was no return of the symptoms.

One of the cases in whom, on July 31st, I opened a knee-joint before the American Congress illustrates a point of interest, for everybody was certain that the man had a torn semilunar cartilage on the inner side of his knee. The internal semilunar was normal and there was no great object in taking a piece out of it. But the case well illustrates the importance of a free incision, for through the large incision on the inner side could be seen a thick flap torn from the external semilunar, so a second incision on the outer side of the patella was made and the affected part of the external semilunar removed. This patient came up to the Out-Patients' recently, forty days from the time he had been operated upon. Two long scars were to be seen, one on each side of the patella, and though the synovial membrane and capsule were only controlled by catgut sutures at the operation, he had a good, sound, firm knee-joint, and was walking about on it just as if he had had only a small incision in one place.

There is no evidence that operation will be followed either by weakness or by stiffness of the joint. The only disadvantages of operation I have seen have been in the knee-joints of elderly men in whom the diagnosis has been suspected rather than made definitely, who have been unsuitable for operation, and in whom the condition of degenerative arthritis of the knee has been accentuated by the operation.

March 15th, 1915.

Tubercle of the cerebellum.—W. Boxwell reports the case of a boy, æt. 10 years, the only child of a father dead of phthisis, who came under observation on January 15th, 1913. There was a somewhat vague history of headache, vomiting, and defective sight. He was a well-developed, intelligent child, and explained that he had had severe headaches at fairly frequent intervals, but that vomiting came on when playing or romping, rather than after food or when at rest. His gait was strutting, with some ataxia, but without tendency to reel to one or other side. The jerks were variable, but generally increased, especially in the right leg, and a clonus could sometimes be developed. The plantar reflexes were also variable. The abdominal reflexes were present, the right cremasteric generally absent. Nystagmus was marked. Distant vision bad. No vertigo, no history of fits, and no tremor. Hearing good. Pupils large and even, tongue normal. On February 1st, after getting out of bed, he became giddy and fell, and about this time he had his first and only attack of vomiting. On February 4th a "lower neuron" paralysis of the left facial nerve set in, followed on February 8th by deafness in the left ear. An ophthalmoscopic examination showed "double-choked disc" with atrophy in the left eye. On February 9th he had an attack of convulsions. The diagnosis of a growth of some sort, probably tuberculous in the left lobe of the cerebellum, was now partly obvious, and the almost sudden development of dysdiadokokinesis of the left arm strengthened the impression. A decompression operation was undertaken. The child never quite recovered from the shock of the operation, and died two days after. A large tuberculous mass was found post-mortem, occupying the left lobe of the cerebellum and displacing the right lobe considerably to the right side. No other evidence of tuberculosis was found.—*British Journal of Children's Diseases*, XI., p. 273.

INDISPENSABLE ORTHOPÆDICS. By F. CALOT. Translated from the sixth French edition by A. H. ROBINSON, M.D., and LOUIS NICOLE. With many illustrations and coloured plates. London: Baillière, Tindall and Cox. 1914. Pp. 1169. Price 21*s.* net.

It is unfortunate that this book, which is so full of the knowledge that only a long practical experience can bring, should have been so literally translated. No attempt has been made to use any English phrase or idiom, and indeed the greater part of the book reads very awkwardly to the English mind. As an example we may quote the "note to fig. 47" which reads: "What it is not necessary to do,"—the figure depicts an error which it is absolutely necessary to avoid. In spite of these defects the book is well worth the serious perusal of all who are engaged in orthopædic work; it is filled with the minute technical detail for which Dr. Calot is so well known. The chapter on the injection treatment of tuberculous glands shows the excellent results to be obtained by a method almost unknown in this country.

ON MEANS FOR THE PROLONGATION OF LIFE. By SIR HERMANN WEBER, M.D., F.R.C.P., Consulting Physician to the German Hospital and the National Hospital for Consumption, Ventnor. Fourth enlarged edition, founded on a lecture delivered before the Royal College of Physicians on December 3rd, 1903. Pp. x + 235. London: John Bale, Sons and Danielsson, Ltd., 1914. Price 4*s.* 6*d.* net.

This book was originally based upon a lecture delivered in 1903 before the College of Physicians. Its general arrangement remains as in former editions, but there are some additions and alterations. The subject is treated from the aspect of prevention and does not enter into the pathological anatomy, clinical symptoms, or mental changes of old age, the author's aim being merely to outline a mode of life by which senile changes may be postponed as long as possible. The main points urged in the sixteen sections of the book are the maintenance of vigour and resisting power by regular daily walks, rides, respiratory and other exercises, with plenty of open air, moderation in appetite and enjoyments, the prevention of disease and the counteracting of any inherited tendency, early hours, daily baths, maintenance of mental vigour by appropriate work, and the cultivation of sympathy, contentedness and will power. The book contains many useful hints and suggestions, as an instance of which may be mentioned the usefulness of regular massage of the thyroid and parathyroid glands and of the larynx to ward off symptoms akin to myxœdema, such as puffiness, falling out of hair, dryness of skin, and mental hebetude. For the last fifty years the author has regularly carried out the rules he lays down, and the fact that he is now in good health in his ninety-first year, while his parents did not pass sixty and most of his forefathers died under seventy-one, is perhaps the best testimony of the soundness of his views. Both the bibliography and index are copious, and the book is printed in good type.

THE INFANT, NUTRITION AND MANAGEMENT. By ERIC PRITCHARD, M.D.Oxon., M.R.C.P. London: Edward Arnold. Price 3*s.* 6*d.* net.

Dr. Pritchard's test of infant feeding is found by weighing the infant before and after. His standard of assimilation is established in this manner. He is, of course, a strong advocate for breast-feeding. It would be interesting to know what percentage of infant cases attended by medical men is due directly and indirectly to dietary indiscretion on the part of ignorant, thoughtless, and feckless mothers. X-rays and bismuth meals are invoked as an aid to diagnosis in malnutrition when motor mechanism of the stomach or pyloric mischief are suspected. Dr. Pritchard insists on training the mother at the expense of the State in infant hygiene, etc. If this were successfully carried out and systematic inspection established, much of the troubles which afflict the infant would disappear.

THE OPERATIVE TREATMENT OF FRACTURES. By SIR W. ARBUTHNOT LANE, BART., M.S., F.R.C.S. Second Edition. London: Adlard & Son. 1914. Pp. 184. Price 10*s.* net.

The new edition of this treatise differs but little from its predecessor of 1905. The author has added numerous radiograms which materially help to demonstrate the operative methods of fixation of bony fragments. Another valuable addition is the section of the report of the Committee appointed by the British Medical Association to report on the ultimate results obtained in the treatment of fractures with or without operation.

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SUSPENSION LARYNGOSCOPY.*

By PROFESSOR GUSTAV KILLIAN.

Translated by D. R. PATERSON, M.D., M.R.C.P., Surgeon, Ear, Nose, and Throat Department, Cardiff Infirmary.

IN 1895 Kirstein saw the interior of the larynx without a mirror for the first time. Notwithstanding the zeal with which he strove to make known and extend the new method, it was a long time before it received general acceptance, and we penetrated by tube spatula into the trachea, and ultimately into the bronchi as far as the eye could see. An immense field was thus opened up.

It seemed hardly possible that anything more was wanted, and I often asked myself, what further? I had forgotten the occasions when, during operation, I felt that better access to the larynx than through a narrow tube was much to be desired. In order to obtain exact pictures of the deeper air-passages I got my artist to work from the cadaver, as time is usually too short to sketch and paint from examination in the living. In the dissecting-room, with the head of the subject hanging over the table and the mouth wide open, a spatula was introduced over the tongue and larynx, and the field of view illuminated by an electric hand-lamp. It was too fatiguing to hold the instrument until the artist had finished, so I fastened it to an iron stand, which was screwed on to the dissecting-table. An altogether new situation, viz. the head of the cadaver suspended on the tongue spatula, was produced. By suitable manipulation, with the mouth as wide as possible, a wonderfully clear view of the bucco-pharyngeal cavity, larynx, and entrance of the œsophagus was obtained (Fig. 1). Many points which have hitherto escaped notice can be learnt by this method. The

* Abstract of the Semon Lecture.

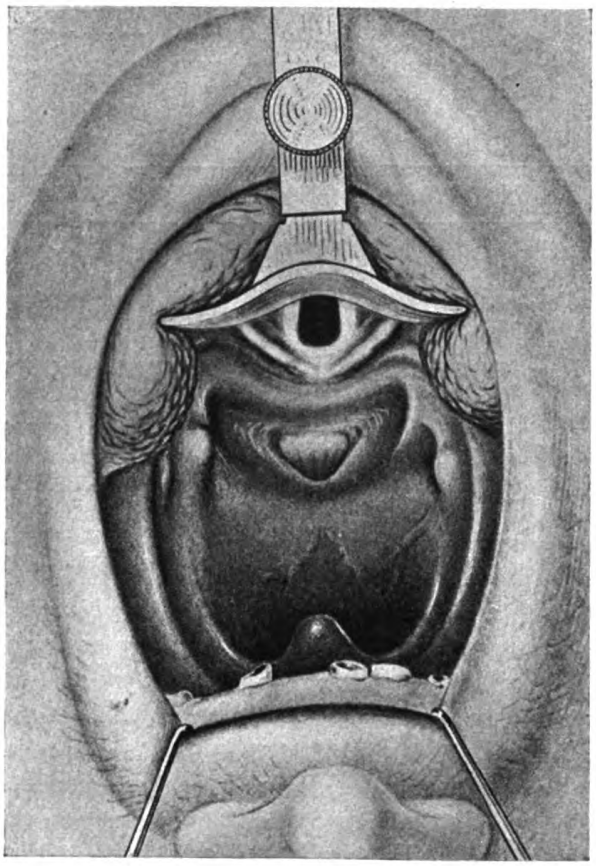

FIG. 1.—View with suspension laryngoscopy in the cadaver. Obtained with the old Kirstein spatula. The epiglottis is hidden by the spatula. Below the epiglottis the hypopharynx and mouth of the gullet are seen.*

posterior pharyngeal wall appears in its whole length and breadth, from the uvula to the mouth of the gullet. Laterally the greater cornua of the hyoid stand out. The posterior surface of the laryngeal cavity is observed in its full extent, as well as part of the posterior tracheal wall. Only the anterior wall of the larynx remains concealed.

The possibility of carrying out the same in the living came into my mind, for thus not only the upper, but also the deeper parts of the pharynx, and especially the interior of the larynx, would be accessible to eye and hand in a manner unsuspected. The situation suggested a comparison with the work of the gynæcologist, who is able, with the speculum in position, to use both hands in operating on deep parts.

The success of the examination in the living appeared to depend upon obtaining the same relaxation of parts as in the cadaver, and this was possible only in deep anæsthesia. I determined, therefore, to undertake the first experiments on anæsthetised patients, in cases where the severity of the disease justified an anæsthetic. The limitation only increased my difficulty, for such cases were altogether too few for the development of a new method. Accordingly the simple spatula hook which I had constructed was used only in three cases—a child with laryngeal papillomata and two cases of tubercle of the larynx. The spatula and the handle for suspension of the head were made out of one piece (Fig. 2), and an excellent result was got with this instrument. The interior of the larnyx was easily accessible and the operation much simplified.

Guided by this experience, further attempts were made with cocaine alone on two patients accustomed to the direct method. In neither was the procedure painful. They remained perfectly still. Suspension laryngoscopy was now frequently performed, and the instruments were so altered as to be suited for general use.

The Tongue Spatula.

Attention was mainly directed to the tongue

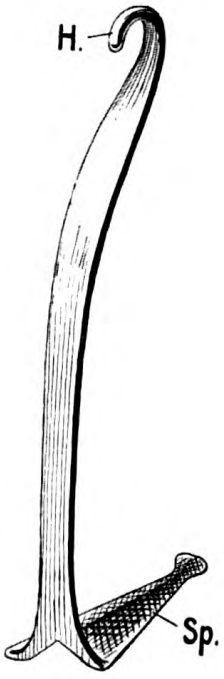

FIG. 2.—The spatula-hook in its earliest form. Made of *one* piece. *H.* Hook. *Sp.* Spatula.

* We are indebted for the illustrations to the *Journ. of Laryngol., Rhinol., and Otol.*

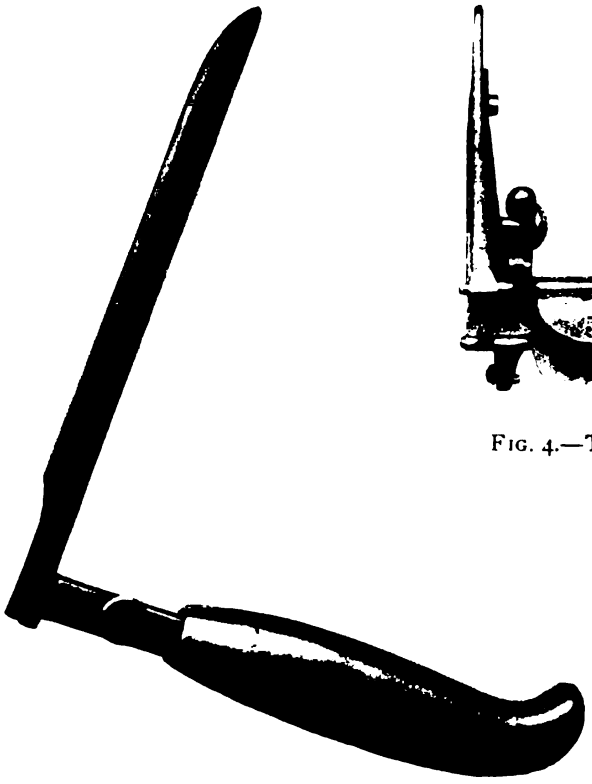

FIG. 3.—The gutter-spatula.

FIG. 4.—The flanged gutter-spatula. Both flanges are adjustable.

spatula and to its handle with the hook-shaped extremity. Since the discovery of direct laryngoscopy, tongue spatulæ of various forms have been constructed. I worked with the tube spatula although I had always made use of the simple long narrow Kirstein's spatula as well. Later I suggested, instead of a complete tube, a split one in the form of a gutter having on cross section the form of a Roman V (Fig. 3). Very firm pressure can be exerted by it on the middle of the tongue, and I have employed it in a somewhat modified form for suspension laryngoscopy from the commencement. It is essential to prevent the tongue bulging too much to the right or left at the side of the spatula and overlapping the gutter. Movable flanges, have, therefore, been fitted to its sides (Fig. 4). The flanges may, by means of levers, be made to assume and retain the desired position. Such a spatula leaves little to be desired in the way of adaptability, and one ought, of course, to have it of different lengths. A second form has also proved of service. Not infrequently it happens that even a well-fixed epiglottis gradually slips from under the spatula and readjustment of the instrument becomes necessary. The epiglottis may be raised easily by means of a small rod, and Albrecht has had a spatula made which can be extended whilst in position. Carrying out this idea, I have attached to the tongue spatula a second narrower and longer one which is inserted through a slot and can be fixed by means of a screw (Figs. 5 and 6). This arrangement enables the epiglottis to be very easily raised. The introduction of the instrument enables in the first instance the tongue up to the epiglottis to be controlled, and in the second place, the epiglottis to be brought out of the field of vision. As spatulæ of different lengths are in use, provision must be made to detach them from the suspension hook when necessary. The shaft of the

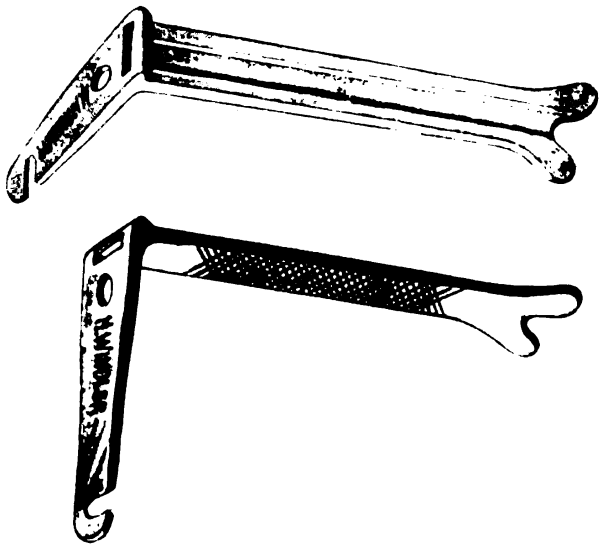

FIG. 5.—Double spatula without epiglottis elevator. Anterior end heart-shaped, behind is a slot through which the epiglottis elevator is inserted.

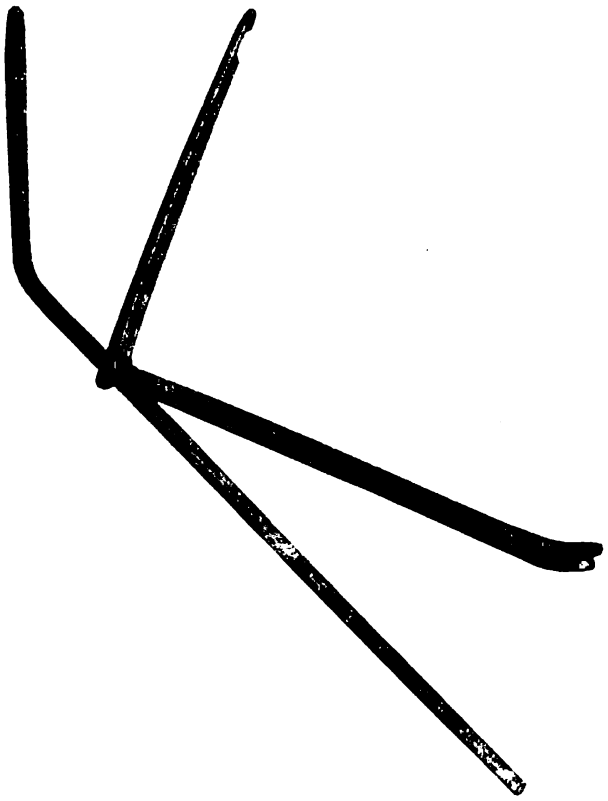

FIG. 6.—Double spatula with epiglottis elevator.

tongue spatula is, therefore, constructed to permit this adjustment.

The Suspension Hook.

The tongue spatula had, as already mentioned, only a straight rod as handle, the end of which was curved in the form of a hook.. Albrecht showed that slipping out of the tongue spatula might be prevented by making the point of suspension fall perpendicularly over the end of the tongue spatula or even in front of it. This is carried out by means of an adjustable joint and a thumb-screw (Fig. 7). On turning the thumb-screw in the direction of the hands of a watch, the hook is inclined towards the tongue spatula and a clearer view of the larynx is rendered possible.

To the part of the suspension-hook below the joint the tongue spatula is attached. It has also a particular arrangement. There is a large screw which comes into play in opening the mouth. The second screw, on its anterior aspect, serves to fix the epiglottis spatula.

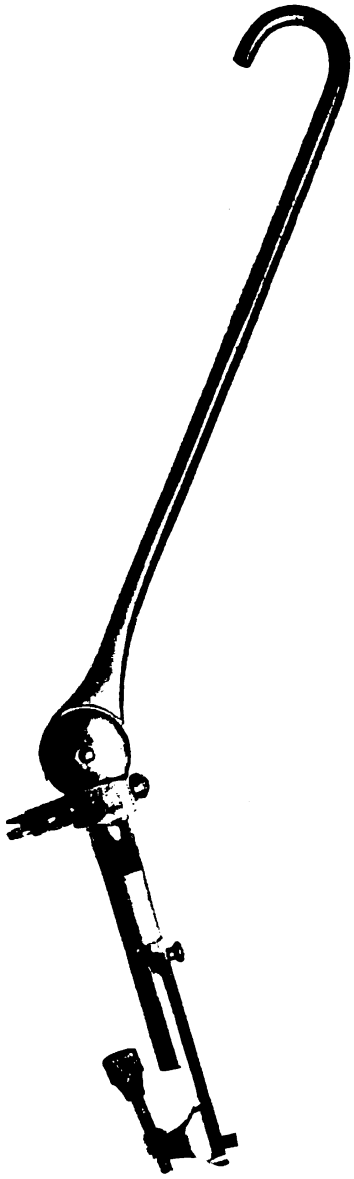

FIG. 7.—The suspension-hook by itself. Joint adjustable by thumb-screw At the lower end screw for fixing epiglottis elevator. Behind, two pins for attachment of tongue spatula.

The Mouth Gag.

As the mouth of the patient must be held continuously open as wide as possible, a gag is absolutely necessary (Fig. 8). This has the form of a bow, which is fitted to the lower part of the spatula-hook, and can be screwed up and down (Fig. 9). The bow impinges by means of a small stop against the incisor teeth, or in an edentulous mouth against the upper edge of the alveolar process. Instead of the stop an adjustable plate can

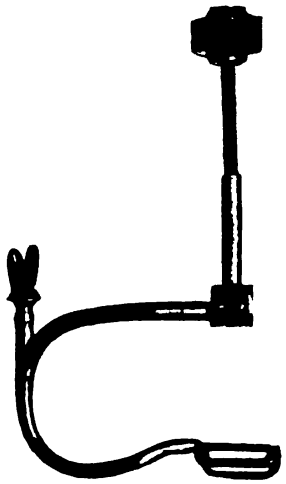

FIG. 8.—The mouth gag. Detached from suspension hook. Lower part of the gag turns on vertical axis and fixed by screw.

be used. Recently the bow itself has been made adjustable in a horizontal direction as well, so that it can be better adapted to the patient (Fig. 10).

The Counter-pressor.

The counter-pressor is intended to exert pressure on the cricoid region of the larynx and to push it towards the tongue spatula (Figs. 11 and 12). It is, therefore, also fixed to the spatula-hook (Fig. 13). I have had it made in the form of a circle, and so arranged the curve that it can be lengthened or shortened. It can be likewise raised up. It presses upon the larynx by means of a plate.

The Gallows.

It is necessary to suspend the spatula-hook, and the point of suspension must be in the position which the hook assumes after the introduction of the spatula. Special apparatus is, therefore, necessary to carry out rapidly and with ease all forms of adjustment, higher, lower, backwards, forwards. This apparatus, which I have called the gallows, is screwed to the operating table (Fig. 14). It consists essentially of an upright carrying a horizontal arm. The upright is attached to the extreme corner of the top of the operating table. It can be placed higher or lower, and by means of a crank-handle to move backwards or forwards. From the upright projects a horizontal arm, which reaches to the middle of the operating table, and can itself be fixed higher or lower. The adjustment of the upright gives the rough, that of the horizontal arm the more detailed, view.

The Operating Table.

Most operating tables are so constructed that the gallows can be attached. It was clear, however, that for suspension laryngoscopy they might be materially improved. The requirements are similar to those for the direct method on patients, in the back or side position.

The first desideratum is to be able to make the

FIG. 9.—The suspension-hook with mouth-gag.

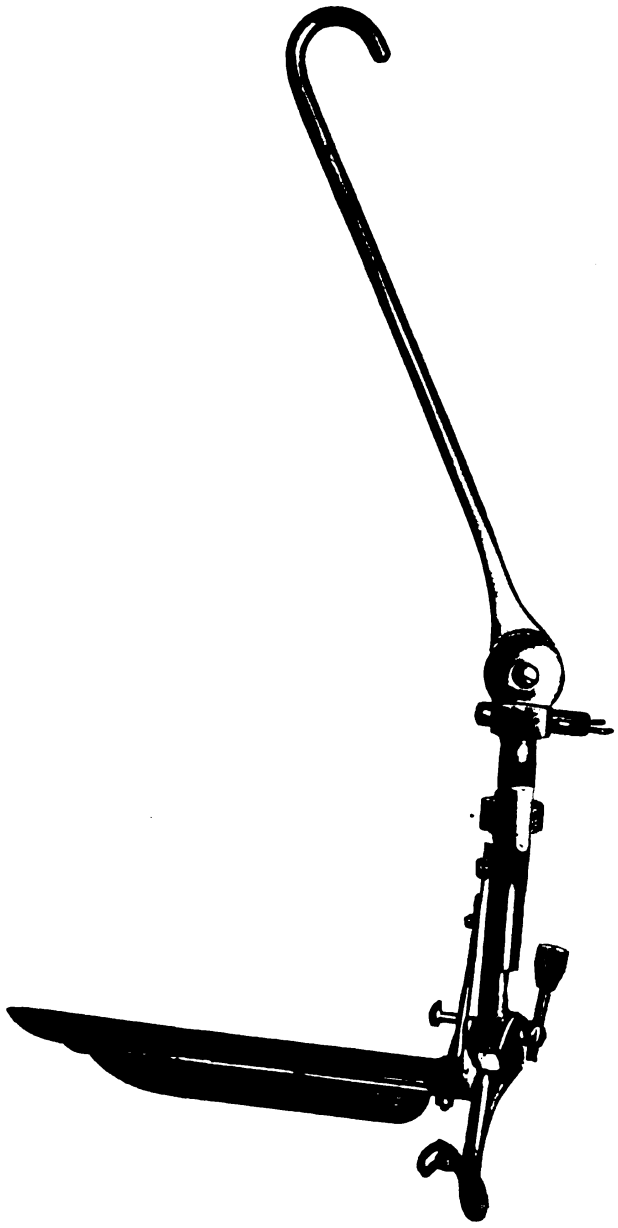

FIG. 10.—The suspension-hook with mouth gag and flanged gutter-spatula, seen from the side.

top of the table higher or lower. With the ordinary table the operator is usually obliged to squat upon a stool or even to kneel. Ordinary operating tables can be raised but little. They do not, as a rule, allow of the operator sitting upon a chair or standing, which is very convenient for demonstration. Kahler has had a special stool constructed, which may be screwed up as high as is necessary. I have had a new table so arranged that it can be raised unusually high (Figs. 15 and 16). This is done by means of a crank-handle manipulated by the operator himself. The top of the table can also be lowered and the back-rest adjusted. For the head a support adjusted by a handle has lately been introduced, so that the help of an assistant to hold the head is no longer necessary.

All operations usually performed by laryngologists may be done on this table, and in this respect it is not inferior to others.

Other Instruments.

For the protection of the operator from expectoration, especially in tuberculosis, a sheet of glass has been fixed to the gallows, so as to come between the surgeon and the patient. Single and double curettes of suitable form and length are necessary for various operative procedures, and so also are galvanocautery points for deep puncture. For hæmorrhage, instruments are made which permit the fastening and removal of clips in a manner similar to those used for uniting skin wounds or controlling tonsil bleeding. Blumenfeld first recommended the use of such clips in the larynx.

ILLUMINATION.

Good illumination is, of course, required. It is possible, with a head-mirror and a good lamp fixed to a stand or held by an assistant, to obtain

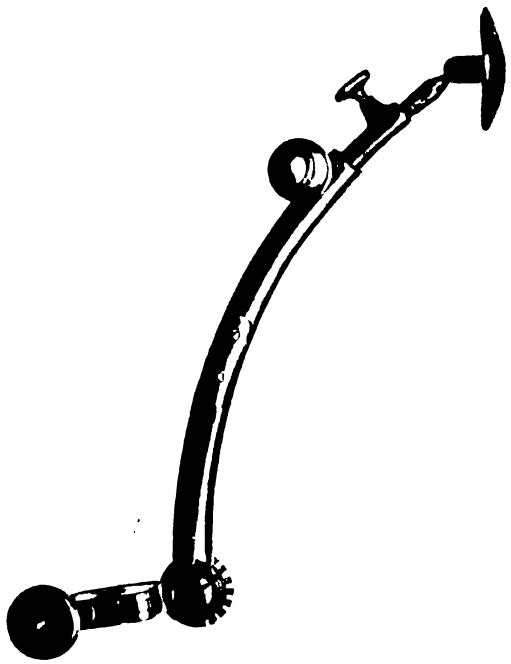

FIG. 11.—The counter-pressor. Closed.

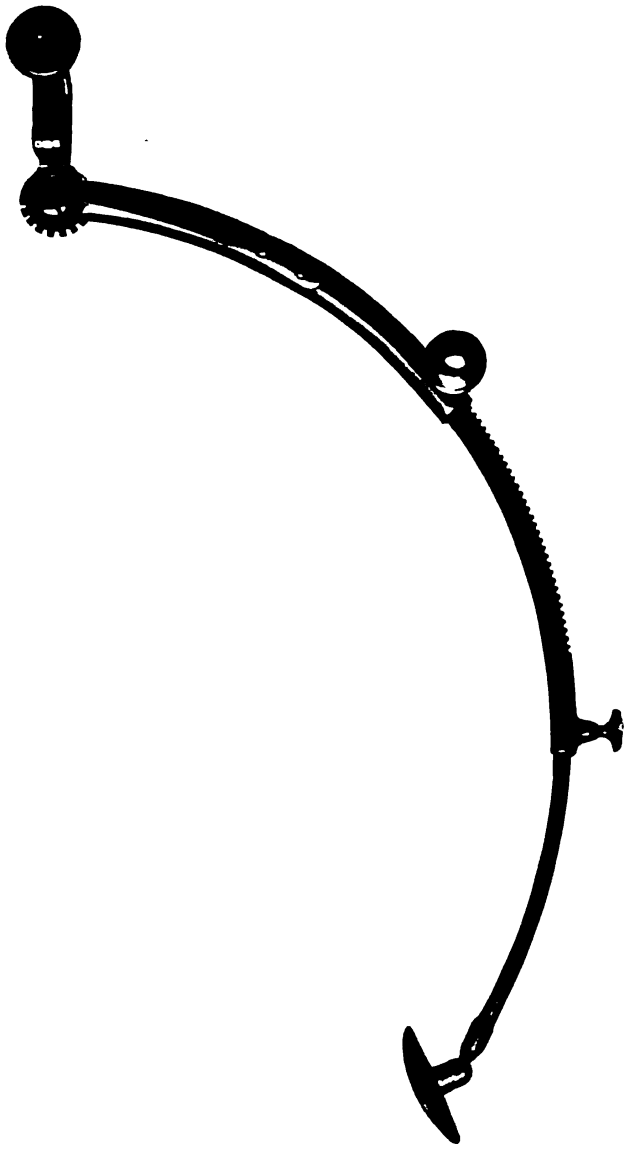

FIG. 12.—The counter-pressor. Extended. Anteriorly the plate which is placed on the cricoid cartilage. At the other end the attachment-mechanism and joint.

sufficient light. The Nernst lamp, with the light concentrated by a lens, is quite suitable. I have always found very convenient the old Kirtstein head-lamp, used as in other operations. Its drawback is that it is not aseptic in construction. I have lately had made an aseptic head-lamp, with a number of innovations. There is a new form of joint which greatly facilitates focussing. The electric lamp has three crossed metallic filament-spirals giving a very strong light. The focussing lens can be adjusted by means of a screw. There is also an iris diaphragm to make the light circle larger or smaller.

Quite new is the employment of a transformer suspended from the roof of the operating theatre. The rheostat swings by a cable free over the operating table so that the flexible no longer drags on the floor, and, being covered with rubber tubing, it can be washed. The head-lamp and the rheostat are balanced by counterpoise, and one can work comfortably with the lamp on the head without the flexible dragging.

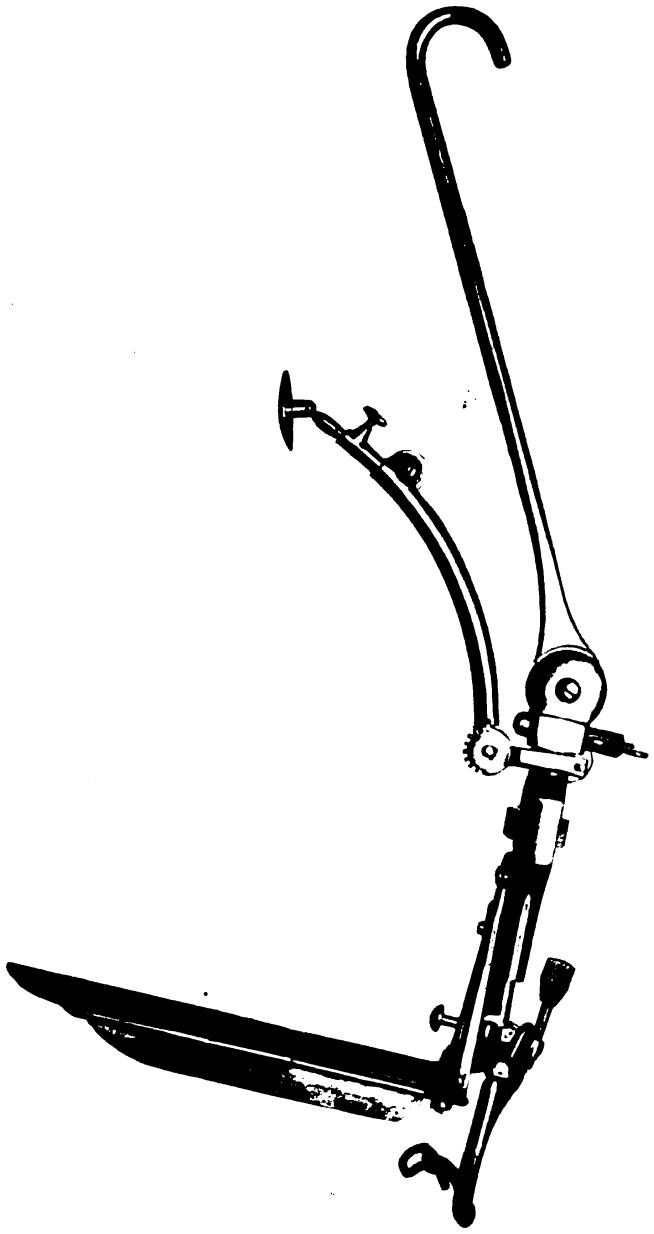

FIG. 13.—The suspension-hook with spatula and counter-pressor. Counter-pressor raised up.

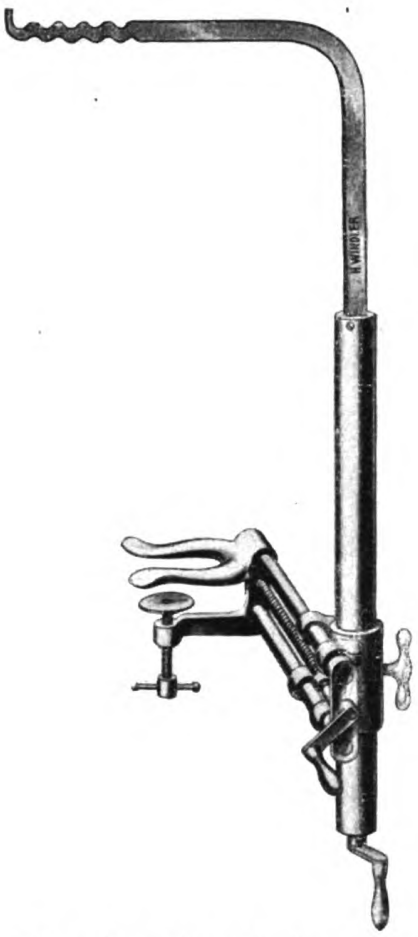

FIG. 14.—The gallows with mechanism for attachment to the operating table.

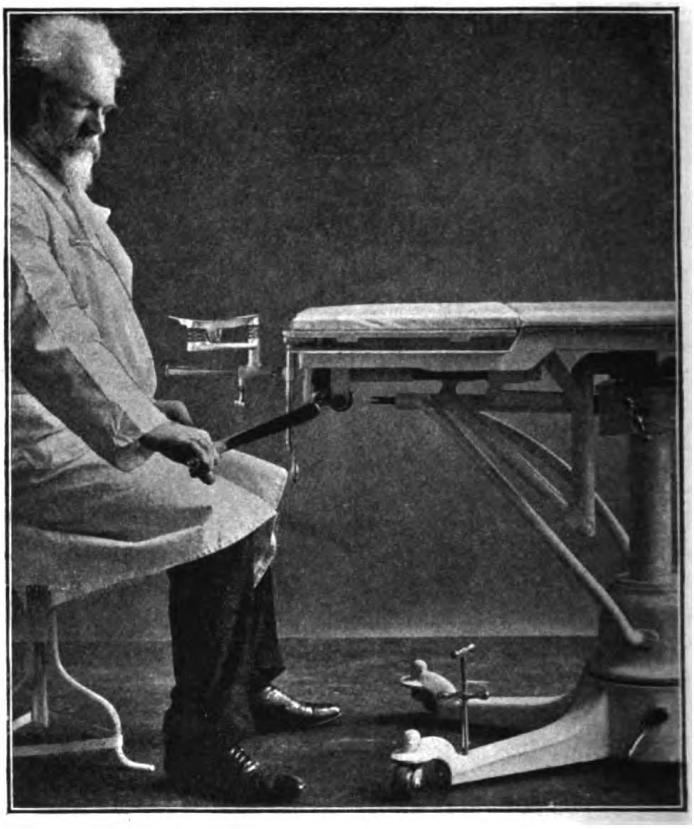

FIG. 15.—Operating table lowered. I am holding the crank-handle which regulates the table top.

For demonstration purposes it is of advantage to have illumination by means of small electric lamps fastened either to the suspension hook itself or to its bow outside. Very effective is the method of having miniature lamps fixed on the end of the tongue spatula by a special apparatus.

PREPARATION OF THE PATIENT.

All patients are not suitable for this procedure. Those who place difficulties in the way of introduction of tubes conduct themselves in a similar manner in suspension laryngoscopy. I refer to patients who do not open the mouth well, in whom the teeth in the upper jaw are very prominent, the tongue very thick and unyielding, and the larynx reached only with difficulty by a narrow tube from the angle of the mouth. In such circumstances it is hardly worth while to attempt the new method.

Before the patient is submitted to suspension laryngoscopy one ought to satisfy oneself as to his behaviour under direct examination. As a rule I introduce beforehand, with the patient sitting and under reflected light, a long tongue spatula, and find out how much the base of the tongue can be depressed. At the same time this examination is utilised to determine the length of the spatula to be used in suspension laryngoscopy. In order to do this accurately Kahler has had Kirstein's spatula graduated in order to measure the distance from the lower incisor to the base of the epiglottis. This is of great advantage.

In children this preliminary examination is unnecessary. Suspension laryngoscopy can be carried out on them with ease. Women also in general stand it well.

The examination is best carried out in the morning with the patient fasting. If the cases are suitable and the patients not apprehensive, endeavour is made to carry it out under cocaine alone. For this I recommend especially the 25 per cent. spirit solution. It is applied to the mucous membrane of the base of the tongue, pharynx and larynx after previous application of the 20 per cent.

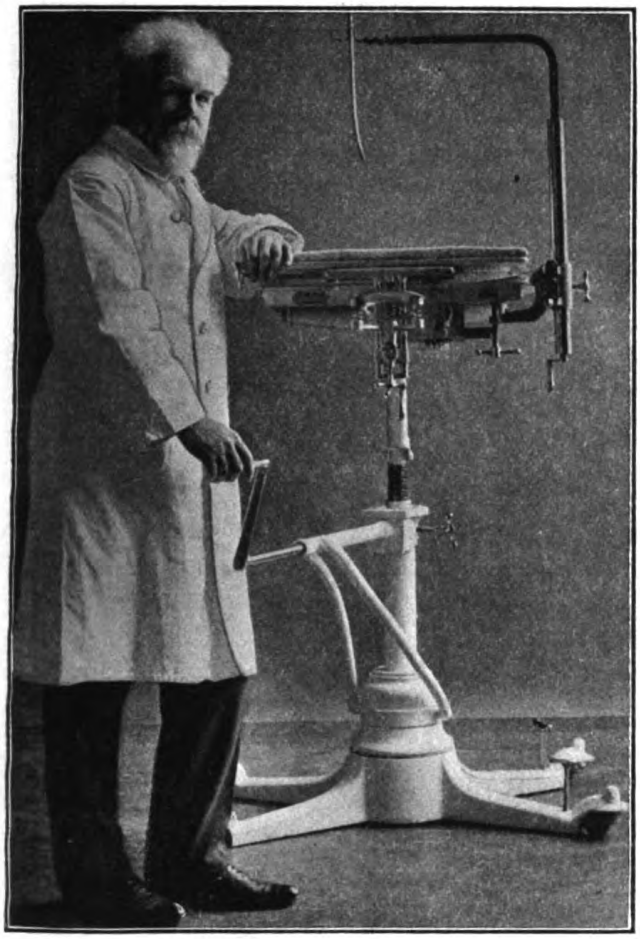

FIG. 16.—Operating table, head aspect. Table raised, gallows clamped on.

aqueous solution. The addition of adrenalin is always helpful.

Children should be put under the influence of chloroform or ether. We begin with a mask and then change to a bellows apparatus, the form devised by Braun being preferred. It is absolutely necessary during and after the introduction of the spatula-hook to blow the anæsthetic under increased pressure, into the deeper air-passages, otherwise with the mouth widely open too little is carried into the lungs and no regular and sufficiently deep anæsthesia is obtained. It is a good plan to give half an hour beforehand according to the age 5 to 10 drops of a 1 per cent. codeine solution in order to lessen cough reflex.

Morphine-Scopolamine Narcosis.

Two hours before suspension the patient is given a centigramme of morphine and three decimilligrammes of scopolamine. An hour later the same quantity of both substances is injected. The operation room is darkened and quiet obtained so that the patient may fall asleep. It is not always successful with this dose, but deep sleep is not altogether necessary. The patient stands the operation quite well even if he is merely drowsy. One can carry on a conversation with him and give directions which he readily follows.

I would point out that with scopolamine and morphine cocainisation of the larynx is by no means superfluous. As a rule, the cocaine brush should be passed several times into the larynx, especially in the tuberculous. I generally have the patient lying on the operating table with the head moderately bent back, and using the electric frontal mirror, I brush the larynx under the guidance of the laryngeal mirror. It is helpful in doing this to have the under jaw pulled forwards as it facilitates the view into the larynx and it is easier to introduce the brush. With morphine and scopolamine it is not necessary to use so much cocaine as both substances materially diminish the sensibility of the larynx. The examination should

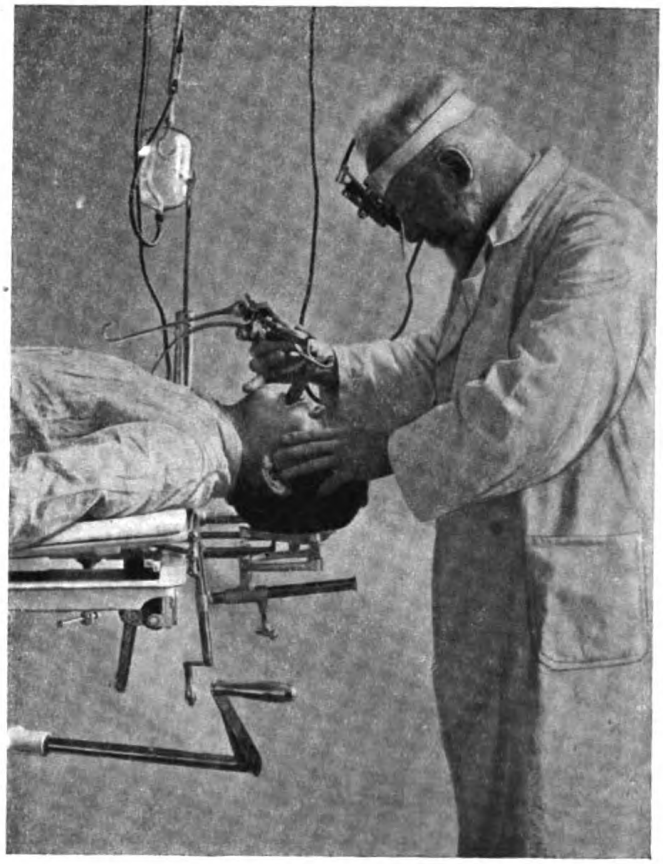

FIG. 17.—Introduction of the suspension-hook.

not be begun in any circumstances earlier than two hours after the first injection, otherwise many patients get into a peculiar condition of excitement and often resist. One can well wait longer—2½ to 3 hours—as the action of morphine-scopolamine lasts for some hours.

Preparation of the Instruments.

Before beginning the examination all the necessary instruments are arranged in order and inspected. The operator ought to make himself sufficiently familiar with the suspension hook and its mechanism. A tongue spatula of suitable length is fitted to the hook and the counter-pressure apparatus fixed above. The gag is adjusted to the minimum distance. Before the introduction the mouth spatula is so arranged that it forms a gutter. An inclination towards the tongue spatula is given to the hook by turning the thumb-screw. It is inclined so that the end of the spatula-hook comes to lie perpendicularly over the point of the tongue spatula. In this form the instrument should be introduced.

The patient lies flat upon the operating table. The body is drawn up so that the head projects free over the edge of the table. It is held in this position by the head support.

The cocainising of the larynx, especially of the epiglottis, and also of the posterior pharyngeal wall and of the tongue, follows. If these parts are sufficiently insensitive, the spatula-hook is taken and the head is depressed somewhat by lowering its support.

Introduction of the Tongue Spatula.

This procedure is easily learned. Those skilled in the practice of the direct method will find no special difficulty. Our task is to pass the spatula under illumination to the posterior pharyngeal wall and then to move it down to catch the epiglottis safely (Fig. 17). The introduction of the spatula is facilitated by pulling out the tongue and holding

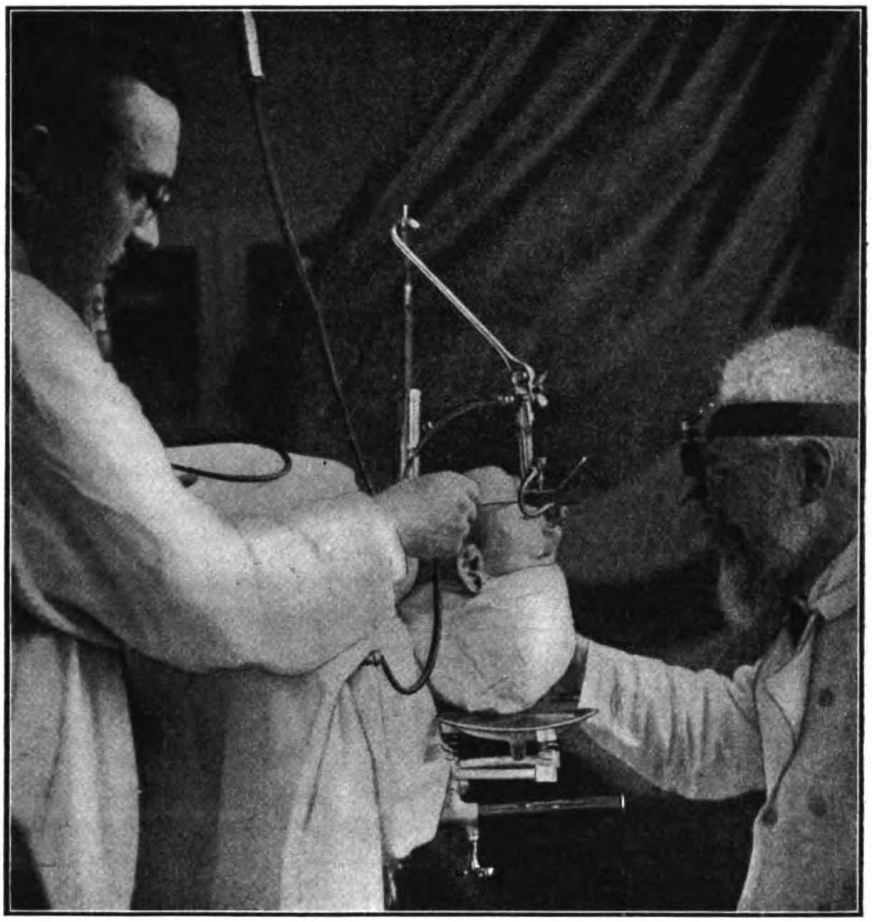

FIG. 18.—Suspension laryngoscopy.

it. This is, however, not absolutely necessary. The tongue spatula readily pushes the tongue downwards if it is not fixed, so that it is well to have it pulled out to enable it to be seized and held.

In introducing the spatula, an endeavour is made to keep exactly to the middle of the tongue. If the epiglottis is seen one glides over it and lifts it upwards. This tilting movement is so made as not to cause the instrument to slip out, its object being to expose to view the arytenoid region and interior of the larynx. If this is successful an assistant must so arrange the gallows that the hook can be suspended from its horizontal arm.

It is only when this stage is reached that the mouth-gag is set in action. Its stop is pushed over the teeth of the upper jaw, the bar fixed and then screwed up as far as the mouth can be opened.

The complete view of the interior of the larynx

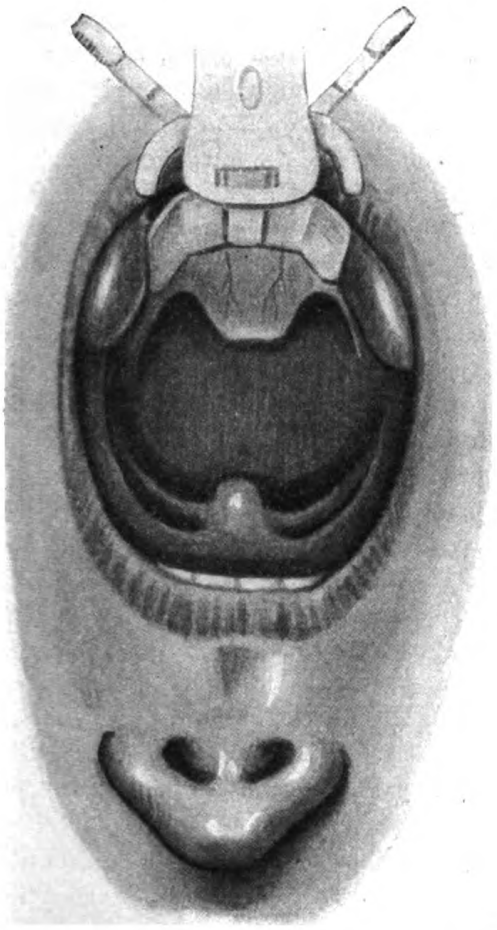

FIG. 19.—The view in suspension laryngoscopy. The epiglottis exposed.

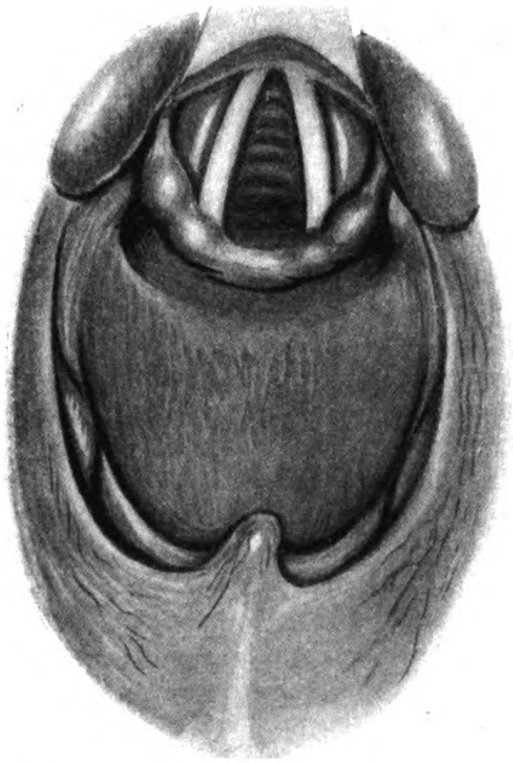

FIG. 20.—The view in suspension laryngoscopy. The larynx exposed.

is possible only when the head support is screwed so far down that the head hangs freely on the tongue spatula. For a more detailed view it is essential to turn the thumb-screw of the spatula hook in the direction of a watch hand. The hook itself will then incline still further towards the spatula.

The anterior commissure usually comes into view only when pressure is made upon the cricoid. We press tentatively with the hand and then apply the counter-pressor. This is accordingly brought down and so lengthened that its plate rests upon the cricoid region. This can be done by turning the screw and making the desired pressure.

If all these manipulations are carried through successfully the interior of the larynx is revealed to view and remains so as long as desired (Fig. 18).

The view of the larynx can be achieved in two movements. The tongue spatula is first introduced over the tongue up to the epiglottis, the hook suspended, the gag put in position, and the head brought into free suspension. This satisfactorily done, the narrow epiglottic elevator is introduced,

pushed well down under the epiglottis and tilted up. The elevator is then fixed with the position screw. If the larynx is in view, the operating table is so adjusted that one can see comfortably, sitting or standing.

The View of the Bucco-pharyngeal Cavity and of the Larynx.

The new method shows the larynx in general, very much as we are accustomed to see it in the direct method. An essential difference is that a complete view is obtained over the whole larynx, pharynx (Figs. 19 and 20), and mouth cavity. The immediate neighbourhood of the larynx projects more plainly, the pyriform sinuses gape, whilst the arytenoid region, and even at times, the beginning of the cricoid plate come forward from the vertebral column. The laryngeal interior appears extended slightly in length in a sagittal direction. The cords are somewhat less freely movable. On deep inspiration a part of the anterior tracheal wall may be seen whilst the epiglottis is covered by the spatula. By lessening the inclination of the head a further extent of the trachea may be seen.

The whole topography of the pharynx and larynx can be taken in simultaneously at a glance, and is intelligible even to a layman. There is no difficulty in the access of instruments to particular parts. Although the intra-laryngeal manipulations demand a certain amount of practice, this is not to be compared with that required for indirect laryngeal work.

Practical Applications.

Demonstration.

The new method lends itself excellently to the demonstration of the interior of the larynx and its pathological alterations to a large circle of students. The demonstrator has practically nothing to do provided the illumination by the small lamp is sufficient. He can stand quietly by and give the necessary explanations. Pathological conditions can be easily seen and correctly interpreted by the students. As specially suited for demonstration I would recommend laryngeal polypi, papillomata, slight cases of tuberculosis, and even recurrent paralysis.

Minor operations can also be shown as, for example, the removal of a polypus or curetting.

Compared with the tiresome demonstrations with the mirror the progress made is very welcome. It is a pleasure to put a probe into the hand of a student and ask him to touch a particular spot with it. If the anæsthesia of the larynx is sufficient, it is always successful.

It has been thought by many that with suspension laryngoscopy operative treatment may pass out of the hands of the laryngologist. I have no such fear. One must be a laryngologist to be able to recognise the correct indications for employing the method. Whoever would use it with success must be familiar with the anatomy, physiology, and pathology of the region as well as with its examination.

Clinical Uses.

Notwithstanding the short time since its introduction, the new method has been used frequently with the best results by a large number of laryngologists in various countries.

From the point of view of diagnosis it is of value, especially in childhood, and, indeed, in all cases where the simple, direct method is used. The only contra-indication is marked dyspnœa. With some practice the introduction of the spatula in children can be done very quickly, and even in difficulty of breathing the larynx can be exposed to view (tracheotomy instruments should, of course, be at hand). With the larynx in sight immediate tracheotomy is no longer necessary in dyspnœa, as a tube of suitable calibre can be passed through the glottis. Artificial respiration can be done with the suspension hook in position.

A whole series of affections of the larynx in children come up for diagnosis, especially where the cause of changes in the voice and breathing has to be determined. There may be revealed a simple acute or chronic catarrh, a subglottic swelling or false membrane, a croupous or diphtheritic affection, node formations on the cords, papillomata, tubercle, syphilis, perichondritis, or a foreign body. It is of value in congenital defects of the larynx and in cases of difficult decanulement.

It gives information on the condition of the hypopharynx. It can be further used in small children in preparation for tracheotomy, bronchoscopy, as well as œsophagoscopy. If the tongue and epiglottis are held up with the spatula, the

tube may be introduced easily and carefully, and this may be repeated without much harm. Before such manipulation the larynx should be slightly cocainised, even where the child is under a general anæsthetic. If this is not done passing the tube is apt to induce a stoppage of breathing from irritation of the pneumogastric nerve, which may be overcome by artificial respiration.

Laryngeal Papillomata in Children.

Of late we have had many opportunities of removing by suspension laryngoscopy recurrent papillomata in small children. From the fact that papillomata often recur even after thorough removal, it frequently happened that the same child had to be repeatedly subjected to suspension laryngoscopy. Although many children had considerable dyspnœa when placed on the table, nothing untoward ever occurred.

The larynx of the child is so accessible in suspension laryngoscopy that the papillomatous masses are readily seen. They can be removed entirely in one sitting and tracheotomy thus avoided, in certain circumstances. For the removal we use a narrow double curette rounded anteriorly, which can be supplemented by the sharp spoon. The larynx is then brushed lightly with a 1 per cent. salicylic acid in spirit. To prevent recurrence administration of iodide of potash and arsenic is of good service.

Recurrence is best prevented by radium or mesothorium. Polyak and I proved that radium caused the disappearance of laryngeal papillomata, and lately, favourable reports of it have come from Chiari's clinic. With the larger quantity of radium or mesothorium now available, papillomata have been caused to disappear. In future they ought to be removed in suspension laryngoscopy and a mesothorium capsule immediately introduced into the larynx.

Vocal Cord Nodes in Children.

In small children who cry a great deal, small nodes not infrequently develop on the vocal cords and lead to persistent hoarseness. They are usually associated with slight chronic catarrh. They are mostly found in children who, in consequence of hypertrophy of the pharyngeal tonsil, thickening of the inferior turbinate, as well as septum deviation, etc., suffer from deficient nasal breathing. The direct method under a general anæsthetic is used for diagnosis. Suspension laryngoscopy is here well adapted, the nodes being easily removed by it.

Tubercle, Syphilis, Difficult Decanulement, etc., in Childhood.

Though tubercle in the larynx is very rare in children we have observed it and in supension laryngoscopy curetted it. No case of syphilis has been recorded.

With regard to difficult decanulement the condition of the subglottic space and of the trachea has to be examined and granulations removed if necessary. For this purpose it may be necessary to pass a tube through the glottis.

In this connection a word may be said on the use of suspension pharyngoscopy. If a broad spatula, which does not extend far down, be attached to the suspension-hook the pharynx is very easily made accessible in small children and operations can be performed, such as tonsillectomy. With the head dependent, the tonsil is seen upside down. Its upper pole is below and the technique of extirpation must be modified accordingly. The blood will run into the naso-pharynx and disturb less. Bleeding may be controlled by compression or by artery forceps and clips.

Foreign Bodies in Children.

Experience of the removal of foreign bodies by means of suspension laryngoscopy from the pharynx of small children is still somewhat limited, but nevertheless very encouraging. Davis appears to have been the first to remove a safety-pin from the pharynx—in a child 11 months old. A piece of bone impacted in the entrance of the larynx and pharynx, a flat bone in the subglottic space in a child æt. 5, and a safety-pin which had lain in the larynx five months, have also been extracted.

An important question is whether suspension laryngoscopy facilitates the introduction of the tube in bronchoscopy in young children. As difficulty is frequently met with, I exposed to view the larynx in two cases of foreign body, cocainised the laryngeal entrance, and then carefully passed the bronchoscopic tube down through the glottis. I succeeded in removing with great ease a metal sheath from the right bronchus. In a similar manner I got from the left bronchus of a two-

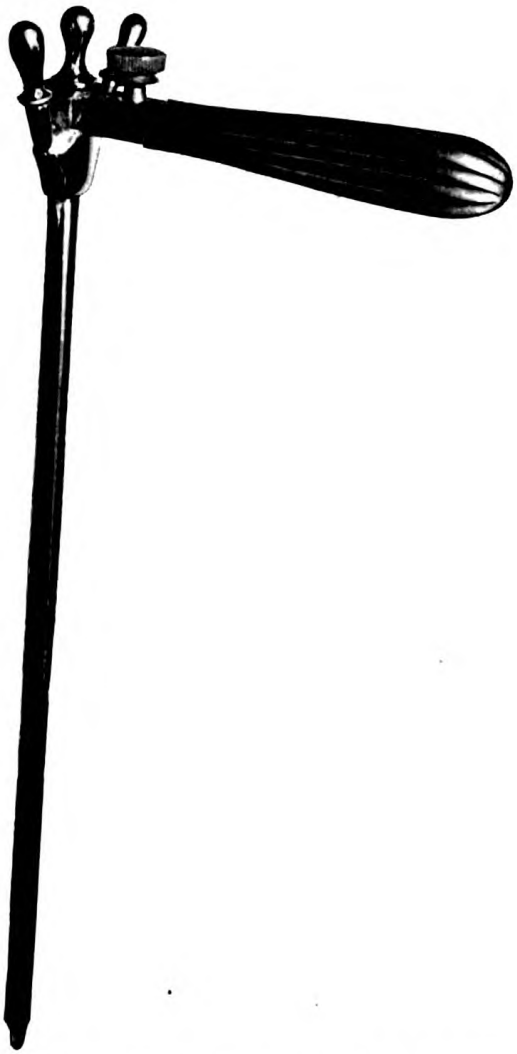

FIG. 21.—New three-channelled tube for bronchoscopy in children.

year-old child a nail which had lodged there for a year and had produced bronchiectatic suppuration. The bronchoscopic tube used on this occasion was of special construction (Fig. 21). It had two fine lateral pipes, one of which extended to the end of the tube, and served for suction of secretions by means of a water-pump ; the second reached only half the length and enabled chloroform to be given continuously during the bronchoscopy, thus keeping up an equable anæsthesia. It greatly facilitated the extraction of the foreign body (Fig. 22).

Suspension Laryngoscopy in Adults. Tuberculosis of the Larynx.

The treatment of laryngeal tuberculosis has at all times given the laryngologist much anxiety and trouble. It was rarely possible to do much because a more vital organ than the larynx, viz. the lung, was generally affected. It was only when we learned to improve the condition of the lung by residence in a sanatorium that better results were obtained in the larynx. Whilst treatment formerly was more or less symptomatic, it later on assumed a more active form. An attempt was made to remove the affected parts in the larynx by operation, or to produce a cure by measures which destroyed the diseased tissue. On account of the great irritability of the phthisical patient this treatment was very troublesome, took up time, and caused the patient much discomfort and suffering. As only very little could be done in one sitting with the single or double curette, it was always an unfulfilled wish to shorten the local treatment as much as possible. I have often felt it would be a great boon to the patient if the major part were done at one time. After-treatment would be materially shortened and sanatorium treatment might be sooner begun.

Since the introduction of direct laryngoscopy operative treatment of the larynx has made marked

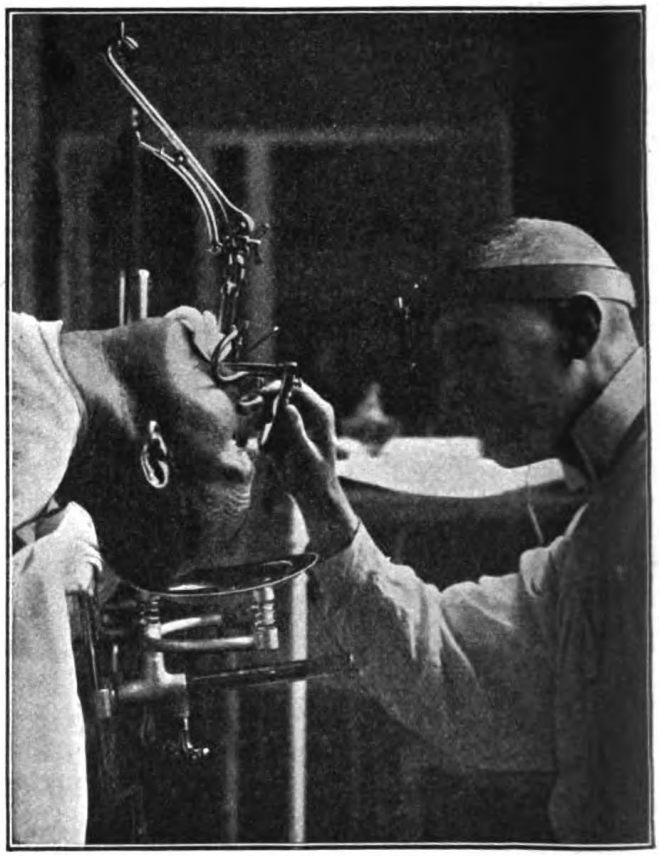

FIG. 22.—Suspension bronchoscopy.

progress. Curettage, as well as the deep puncture by the galvanocautery recommended by Grünwald, could be carried out with the greatest precision through the tube spatula. Something was lacking, however, which has now been supplied by suspension laryngoscopy. It has laid bare to the operator the whole laryngeal region. He can now work with both hands, using the curette and the mop at the same time. Patients tolerate quite well in one sitting an extensive curettage and even deep puncture of an infiltration.

The outlook from the therapeutic point of view is now quite different. Sanatoria are accessible to the poor. For small tuberculous foci sanatorium treatment is sufficient. Definite infiltration, or ulceration, or extensive involvement of the larynx is removed in suspension laryngoscopy by curette or cautery and the patient then transferred to a sanatorium.

With regard to technique, minor manipulations may be carried out forthwith under a morphine injection and cocaine. For more extensive operations, and in very irritable patients, morphine-scopolamine narcosis is well adapted. The operator should have between the patient and himself a glass screen to protect against being coughed upon. The single curette, which can be rotated, is the best; occasionally the double form is required.

With such active treatment I have never met any serious bleeding. If it should happen, the larynx is sufficiently accessible in suspension to apply an astringent or even a clamp. For disinfection of the parts after the operation iodoform or vioform may be insufflated.

This treatment is followed by reaction for a few days. The mucosa becomes injected and œdematous in places. The patient should therefore be kept quiet and forbidden to speak so as to avoid increasing the reaction. In œdema Albrecht's hot-air bath (Fig. 23), which is useful in other laryngeal affections, is excellent. Hot air induces active hyperæmia of the larynx and leads to absorption of the exudation.

It is astonishing how clean the wounds and ulcers in the larynx look some days after the curettage, I have seen nothing like it hitherto, even after prolonged treatment. In favourable cases, the fresh surfaces soon cicatrise under lactic acid applications.

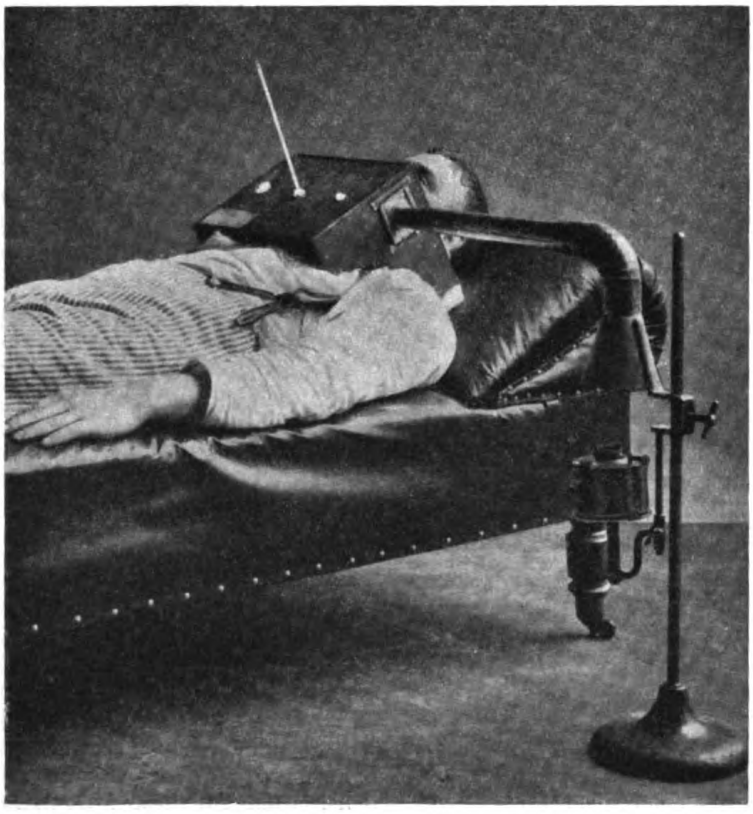

FIG. 23.—Albrecht's hot-air box for the neck.

During the first few days, some pain in swallowing is complained of and there is almost always some rise of temperature similar to what is met in consumptives under the most varied conditions. But it very soon falls. As after-treatment, administration of iodide and the application of hydrogen peroxide, as suggested by Pfannenstiel, give the best results. Only after this should lactic acid be used. An early radical treatment has at times rendered tracheotomy unnecessary. Lupus of the larynx, especially its extensive form, is amenable to treatment by suspension laryngoscopy.

Benign Growths of the Larynx.

Although simple neoplasms are very easily removed under the mirror, suspension laryngoscopy will often prove of value in cases of difficulty. It is especially adapted for extensive growths. Much more can be attempted and carried out with greater precision. At times it will obviate the necessity of an external operation. Removal of a polypus from the vocal cords is extremely simple and even the unpractised may perform it in suspension.

Cancer of the Larynx.

Suspension laryngoscopy ought to render important service in laryngeal cancer. In cases of difficulty removal of a part for examination will be facilitated. Very small growths and suspicious places have hitherto been removed under the mirror or through the tube spatula. Areas on the cords, on the epiglottis or on the aryepiglottic folds can be treated in this way. Suspension laryngoscopy enlarges this field of usefulness, but the limits of intra-laryngeal removal should be very definite. E. Meyer reports excision of a carcinoma from the epiglottis. Such cases, it is needless to say, ought to be selected with care.

In treatment by radium and mesothorium, suspension may present certain advantages for their introduction. I have treated a number of cases with mesothorium and tried various methods. Many tolerate it fairly well if the mesothorium capsule, fixed to a wire or to a slender rod, be introduced into the cocainised larynx and the wire placed between the teeth (Fig. 24). As a rule tolerance for an hour or more presupposes favourable conditions. I have several times introduced the capsule after a morphine-scopolamine injection, the head being lightly supported during the seance, and found the patient able to bear it an hour to

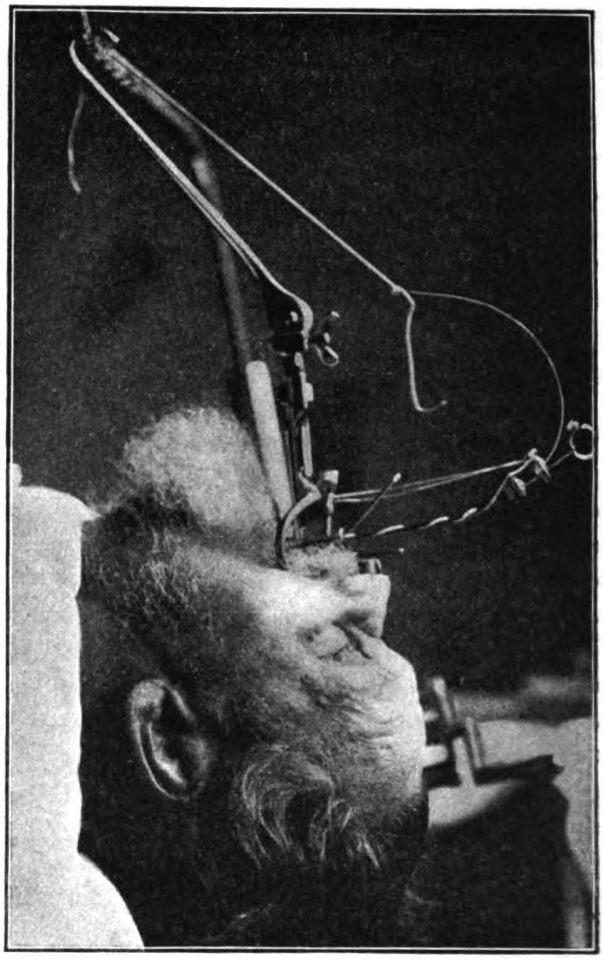

FIG. 24.—Application of mesothorium.

an hour and a half—a much shorter time than is given in gynæcology. The larynx reacted markedly during the first few days after the application, especially if carcinoma were present, and œdema sometimes appeared. It is well to avoid a longer seance than an hour and a half.

A definite effect showed itself in each case. The carcinoma became smaller, but no cure resulted. The growth recurred and the gland metastases were unaffected. Carcinomatous glands should always be removed as they are uninfluenced by mesothorium. Laryngeal cancer, even flat-celled epithelioma, is not nearly so hopeful as carcinoma of the uterus.

New Growths and Foreign Bodies in the Hypopharynx.

The hypopharynx is widened by the lifting up of the larynx and can be more easily dilated with a suitable instrument. Impacted foreign bodies such as dentures can be extracted without difficulty. Œsophagoscopy is not materially facilitated by suspension. Seiffert records a case of removal of a lipoma from the hypopharynx.

March 22nd, 1915.

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We regret that owing to the war the number of pages must be diminished for a time.

A LECTURE

ON

EMPYEMA.*

By J. WALTER CARR, M.D., F.R.C.P.,
Senior Physician to the Royal Free Hospital and to the Victoria Hospital for Children, Chelsea.

GENTLEMEN,—I have, this afternoon, to deal with two diseases, both known by the same name—empyema; the connecting link between them being that in each there is pus in the pleural cavity; but they differ entirely in causation, in course, in symptoms, and, most of all, in prognosis. The one disease is produced by the pneumococcus, and it accounts for the majority of cases of empyema, especially for those occurring in early life. It is nearly always a sequel to acute pneumonia. Occasionally a pleural effusion after pneumonia is not purulent, but that is exceptional—in children very exceptional indeed.

The other disease is produced by the streptococcus, occasionally by that closely associated microbe the staphylococcus, sometimes by the two combined. This form of empyema occurs mostly in adults, usually as part of a general septic or pyemic process; sometimes in connection with a virulent form of one of the specific fevers, especially scarlet fever; sometimes by extension from the lung in cases of septic infarct or gangrene, or from the abdomen by extension from sub-phrenic abscess, appendicitis, or puerperal peritonitis. Not uncommonly it is met with in association with osteomyelitis, and then nearly always in young children. It may be started sometimes as a result of infection by a septic wound of the pleura.

The question may be asked, "What about empyema produced by the tubercle bacillus?" Briefly, I would say that—contrary, perhaps, to the

* Delivered at the Polyclinic.

general impression—it hardly exists. We know that the tubercle bacillus, like the so-called rheumatic diplococcus, is a very rare cause of purulent inflammation. It may, however, be responsible for the production of an empyema indirectly in the following way: Pulmonary tuberculosis gives rise, not uncommonly, to pneumothorax, and we know that in a considerable proportion of cases of pneumothorax, pus forms after a time in the pleural cavity, giving rise to the condition called pyo-pneumothorax. In these cases, however, the formation of pus is not due to the tubercle bacillus, but to a superadded infection, usually by the streptococcus. Nearly all chronic tuberculous cavities in the lung become infected by streptococci. Naturally, when such a vomica ruptures into the pleural cavity, with the formation of a pneumothorax, a streptococcal infection of the pleura follows and an empyema results. On the other hand, when a pneumothorax occurs in connection with a very early case of tuberculosis of the lung an empyema rarely develops, for in such a case there is no question of infection by streptococci or other pus-producing microbes.

Symptoms.

First of the pneumococcal form. It almost always follows acute pneumonia, especially in children, but there is no necessary relationship between the severity of the precedent pneumonia and the tendency to subsequent formation of an empyema. Possibly it is rather more likely to occur if the pneumonia has been severe, but it is also met with quite frequently after perfectly mild cases. Sometimes the empyema starts before the attack of pneumonia has come to an and, and then there is no break in the fever—an important point to bear in mind; we find simply that the more continuous fever of the pneumonia gradually passes into the more remittent fever of the empyema. Sometimes there is a definite crisis, followed almost at once by another rise of temperature due to the formation of pus. Sometimes several days elapse—even seven or eight—of perfectly normal temperature, and then the supervention of an intermittent type of fever indicates the commencement of an empyema. It is important to remember how long the interval may be, especially in children. I recollect the case of a small boy who had had acute pneumonia. After the crisis he apparently made a perfectly satisfactory convalescence, the physical signs cleared up, and he was about to leave the hospital. A few hours before he was to be discharged his temperature suddenly rose and an empyema rapidly developed. So remember, as a definite rule, that we are not safe from the possibility of the development of an empyema after pneumonia until at least a week has elapsed since the crisis. When pus begins to form, it often collects with astonishing rapidity, and may fill the pleural cavity in two or three days. There is usually more or less wasting, loss of appetite, pallor, perhaps a yellowish tint of the skin, and general malaise. The most important fact, however, to note, as far as diagnosis is concerned, is that the symptoms may be extremely slight, and especially, that there may be little or no fever. We often find it difficult to realise that pus may form in large quantity in the interior of the body, as in the pleural cavity, with very little if any rise of temperature. In some cases of pneumococcal empyema, especially of non-virulent type—and the virulence of these cases varies very much—the only indication of what is happening may be that there are persistent signs of fluid at one base, and the child evidently is "not getting on," is not making that rapid and satisfactory convalescence which we normally meet with in a child who has had an attack of acute pneumonia.

The symptoms of the streptococcal form are usually very different. There is severe constitutional disturbance, frequently rigors, and a markedly hectic temperature. Occasionally, in very bad cases, the temperature is subnormal, an indication of greatly diminished resistive power. We are not likely, however, in such patients to be misled by the absence of fever, because the patient is obviously extremely ill, whereas in the pneumococcal form when there is but little rise of temperature the patient may hardly seem ill at all. There is, in the form I am now describing, a marked and rapidly progressive cachexia, with wasting, diarrhœa, profuse sweats, and prostration, so that the patient may rapidly pass into what is known as "the typhoid state." The disease, if not treated suitably, rapidly proves fatal. Sometimes in the worst cases the pus is fœtid.

We may note that in both varieties of empyema the purely respiratory symptoms are, as a rule, very slightly marked; no pain, no cough, no expectoration. Perhaps there is slight dyspnœa, but as long

as the patient is lying quietly in bed it may be scarcely noticeable.

Physical Signs.

These are the same in both classes of case, and are simply those of a pleural effusion. I need not dwell upon them at any length; there is diminished vocal fremitus, dulness on percussion, with markedly increased resistance; as a rule, diminished or absent breath sounds with diminished vocal resonance, and—a most important point in most cases—some displacement of the heart.

A very important question is: Are there any physical signs which indicate that we are dealing with a purulent effusion, rather than with a simple inflammatory one? The reply, unfortunately, must be that in the great majority of cases there are no distinctive signs whatsoever. Very occasionally we meet with signs which are characteristic; for instance, there is what is known as "pulsating empyema"; the pulsation being communicated to the empyema by the heart beats. It is only met with on the left side, and when the left pleura is very full of pus, and even under these circumstances is extremely rare. It is an interesting phenomenon, but so exceptional that we must not attach any real importance to it. Now and then we meet with some bulging of the intercostal spaces over the empyema. Note that this rarely, if ever, occurs as the result of a simple pleural effusion. A purulent effusion exercises more pressure, and, as the inflammation is more intense, it extends to the structures covering the intercostal spaces, and weakens their resistance, so that bulging more readily follows. Lastly, we occasionally see actual œdema of the chest-wall over the empyema. Now and then, I regret to say, we still meet with a pointing empyema. Of course, when this occurs there is not much doubt as to the nature of the case, but the diagnosis should be made long before that stage has been reached.

If an empyema is left untreated many dangers follow. It commonly ruptures, sooner or later, either externally or internally. Externally, it may rupture practically anywhere on the surface of the chest; but most frequently in the fifth intercostal space, at about the nipple line, because there the muscular covering of the chest-wall is about at its thinnest. There is, first of all, bulging and eventually rupture. If rupture occurs internally, it is most frequently into the lung itself, and the consequences are very various. Sometimes pus is discharged in large quantity into the lung, and sudden death may follow. Fortunately, it is more common for the patient to cough up the pus and after a few days of very severe illness, improvement sets in, all physical signs gradually clear up and complete recovery ensues. I have seen several such patients come into hospital looking extremely ill, coughing up large quantities of pus, and with signs of fluid at one base; day by day they have improved, without any special treatment, and in a few weeks have got quite well. Unfortunately, in many cases the results are not so good, for sometimes the lung undergoes disorganisation, septic changes are set up, and the patient steadily goes to the bad, with a hectic temperature and increasing constitutional symptoms. An empyema, however, may rupture internally in other directions. It may pass down behind the diaphragm, and burst eventually into the peritoneal cavity or the stomach; or it may appear in the back as a lumbar abscess, or further down as a psoas abscess. I believe there is one case on record of an empyema which gradually tracked down and pointed at the heel! A further danger to be remembered in connection with an old-standing empyema is its very curious association with cerebral abscess. We know that such an abscess may occur as a secondary consequence of any old suppurative mischief in connection with the chest, except fortunately with tuberculous disease. It may follow upon bronchiectiasis, empyema, or abscess in the lung. It is a most curious association and one which can hardly be satisfactorily explained. Doubtless, it is a pyæmic manifestation, but why the abscess should appear in the brain, and not in any other part of the body, is most difficult to say.

Diagnosis.

Obviously this is a matter of the greatest importance. There are some diseases in connection with which it must be said that when we have diagnosed them we can do very little further; so, after all, it does not so much matter whether we make a correct diagnosis or not. This is the case, for instance, with an inoperable cancer in the interior of the body, or, as an example of a different kind, with acute pericarditis. It is certainly only right that

we should make every effort to arrive at a correct diagnosis, but, unfortunately, when we have done so it makes exceedingly little difference to the treatment. With an empyema, however, the matter is very different; it calls at once for active treatment, for the longer pus is allowed to remain in the pleural cavity the greater are the risks of perforation in one direction or another; the greater also the danger that when the abscess is opened the lung will not satisfactorily re-expand. The pus exercises great pressure on the lung, and that organ is apt to become bound down by dense firm adhesions, so that even after the abscess has been drained it may be impossible for the lung to re-expand, and a very unsatisfactory condition of affairs is left.

There are many possibilities of error in connection with the diagnosis of empyema. First, in regard to the pneumococcal form. This, I think, is sometimes missed because of what may be termed "the obsession of unresolved pneumonia." The diagnosis of pneumonia has been made, quite correctly; the temperature never comes down, but merely alters somewhat in type; the physical signs, as I shall explain directly, may be rather misleading; and the empyema may be very small, so that the result is that the possibility of its occurrence is hardly thought of. All experience in medicine shows that if we do not think of a disease we are not very likely to diagnose it when we come across it. If we remember that there is always a possibility, after acute pneumonia, of the development of an empyema we shall not often miss it when it does occur. The streptococcal form is sometimes overlooked because the constitutional symptoms so markedly predominate over those connected with the chest. Also the patient is obviously so ill that it may be very difficult to make a satisfactory physical examination, and the consequence is that the development of an empyema goes unsuspected.

Remember that there is only one certain way of making sure of the presence of an empyema, and that is by exploration. Directly we have any suspicion that pus may be present in the pleural cavity our duty is at once to use the exploring syringe. Do not rely upon anything else whatsoever, either symptoms or physical signs. I have already pointed out that there are no reliable distinctions in physical signs between a simple and a purulent pleural effusion; and the same may be said of the symptoms. A simple pleurisy may give rise to general constitutional symptoms exactly similar to those of a purulent one. Two patients may be lying in adjacent beds, their temperature charts may be practically identical, but the one patient may be suffering from a purulent and the other from a simple pleurisy. Occasionally, for a time at any rate, the symptoms of a case of ordinary pleurisy may be more marked and severe than in a patient with empyema.

In the diagnosis of pleural effusions, both simple and purulent, but especially in the latter, the auscultatory signs may be actually misleading rather than helpful, so much so that we should often do better if we did not auscultate at all, but relied entirely upon percussion and evidence of displacement of the heart. The reason is that over fluid, and especially over pus, it not uncommonly happens, particularly in children, that we hear well-marked bronchial breathing and bronchophony. The result is we conclude that we have to deal with consolidation rather than fluid, and that the case is therefore one of unresolved pneumonia. We overlook the significance of the persistent temperature and of the stony dulness, a dulness more marked, and especially more resistant, than that of a pneumonic consolidation. It is sometimes said that a leucocyte count will help in the diagnosis of pus. All I can say is, Don't rely upon it for a moment, especially remembering that there is, almost invariably, a high leucocyte count as the result of pneumonia, and if, later on, it remains high we have no means of saying whether the excess is still due to the pneumonia or to the subsequent formation of an empyema. So, let me repeat, let it be an invariable rule to explore in the dull area if, after an attack of acute pneumonia, especially in a child, the physical signs —never mind exactly what they are—do not clear up, and the patient is obviously not getting on.

This leads me to speak of certain precautions and details in connection with the exploration. I need hardly mention the necessity for the strictest possible antisepsis—that goes without saying. If the effusion is a large one it does not much matter where you explore, you are pretty sure to strike pus; but in many cases—I am speaking now especially of the pneumococcal ones—the empyema is very small, containing perhaps only two or three drachms of pus. In such a case the area of dulness is likely to be quite limited, and it is most important to

percuss it out carefully, and then explore in the centre, because dulness extends a good deal beyond the limits of the actual effusion. I have known small collections of pus missed through failure to attend to this precaution—through being content to insert the needle anywhere where there is dulness.

Secondly, remember that the chest-wall of a small child is very thin, and consequently you do not want to introduce the needle deeply, otherwise —especially in the case of a small empyema—it is easy to pass it right through the pus into the lung beneath. On the other hand, a small empyema may have a very thick hard wall, which may need a good deal of force to penetrate.

Thirdly, always use a large needle, because in these cases the pus is often rather thick and a needle of small calibre may at once get blocked with flakes of fibrin, so that no pus enters the syringe. Bearing in mind this possibility, when you withdraw the needle, in a case of unsuccessful exploration, be careful to expel its contents into a dish and examine them carefully, because you may find a droplet of pus in the needle itself, which may be sufficient to establish the diagnosis.

Finally note that there are some cases of special difficulty. Occasionally we meet with what is known as an inter-lobar empyema, that is a collection of pus between two lobes of the lung, or sometimes with a sub-diaphragmatic empyema, a collection between the lung and the diaphragm. Such a localised accumulation may be very difficult indeed to detect; in fact, if you do not succeed, after one or two explorations, in finding pus and yet you are convinced from the symptoms and the physical signs that it is probably present, it is best to anæsthetise the patient and make a series of explorations of the chest, beginning, if necessary, in the first intercostal space, and working steadily downwards. I have known pus found in this way when repeated ordinary explorations have failed. Note, however, that a localised empyema at the apex is excessively rare.

In some doubtful cases an X-ray examination is distinctly helpful, and may not only indicate at once the position of a localised empyema, but may serve also to distinguish between an empyema and a subdiaphragmatic abscess, a diagnosis which it may otherwise be very difficult indeed to make, as pus beneath the diaphragm and pressing upwards gives rise to very similar physical signs and symptoms to pus situated immediately above the diaphragm. The X-rays, too, may distinguish between an empyema and an unresolved pneumonia, a comparatively rare condition, but one occasionally met with. I saw a case of this kind a few months ago. A girl, æt. 5 years, was admitted to hospital suffering from double pneumonia. For several days she was extremely ill, but the temperature fell to normal about the twelfth day. Very soon, however, it rose again, and for nearly four weeks ran an irregular course, varying between 98° and 103° F. At the same time the patient had signs strongly suggestive of a right pleural effusion, except that the heart was not displaced. She also had a very persistently high leucocyte count, about 40,000 leucocytes per cm. In fact the history, physical signs, symptoms and blood examination pointed most definitely to the presence of an empyema; there seemed no other possible diagnosis. The chest was explored repeatedly, but always with a negative result. Finally an X-ray examination was made and showed that there was no collection of fluid, but merely a general diffuse opacity over the greater part of the right lung, with some entry of air everywhere—undoubtedly a case of unresolved pneumonia. Eventually the temperature gradually fell to normal and the patient got quite well. In such a case an X-ray examination may save a number of unnecessary exploratory punctures, and this, especially in the case of a small child, may be important.

It is very easy, when an empyema is present, to overlook a purulent pericarditis; and the two are not uncommonly associated. It is easy to see how the physical signs of the one condition may mask those of the other. Fortunately such an error is of no great importance, because purulent pericarditis is almost necessarily fatal.

Prognosis.

There are three chief factors to consider. First, as already explained, the nature of the causal microbe. Pneumococcal empyemas, if treated promptly, commonly do well, especially in children. The lung usually re-expands, and even if it does not the soft chest-wall readily falls in to some extent. Drainage rarely needs to be maintained for more than a week or two, and complete recovery is the rule. Streptococcal and staphylococcal

empyemas mostly do badly; the disease is of a much more virulent type and the symptoms of a graver character; moreover, as already explained, there is always the possibility, or the probability, that the empyema is merely part of a general septic infection.

Second, the age of the patient; to this I have already referred. Children do better than adults, for two reasons: First, because the empyema is usually pneumococcal; secondly, because the lung expands so much more satisfactorily and the chest-wall falls in more easily.

Third, the length of time which has elapsed before the empyema is drained. An untreated empyema tends, of course, to reduce the patient's vitality; moreover, the longer it is left the greater are the risks of serious complications arising, and also the less likely is the lung to expand. If the lung does not expand a large pus-secreting cavity remains, which is unable to contract to any appreciable extent, especially in the rigid chest of an adult. It is extremely difficult, in fact almost impossible, to keep such a cavity aseptic for any length of time. Sooner or later it becomes septic, and then we get increasing risks of the development of lardaceous disease, the patient gradually losing ground, and finally succumbing to the activity of the septic process. Also, as already mentioned, there is the ever-present danger of the development of a cerebral abscess, which always proves fatal in a very few weeks.

Treatment.

This is essentially surgical and I need not dwell on it. There is only one thing to do for all cases of empyema, whether pneumococcal or streptococcal—to open and drain them at the earliest possible moment. There is just the possibility that a pneumococcal empyema may dry up. A generation or so ago, it was the custom to aspirate an empyema, at any rate a pneumococcal one, once, perhaps twice, and then, if it re-filled, to open and drain. It happened very occasionally that the pus did not re-accumulate and the patient was cured. Nowadays we recognise that the probability of such an occurrence is so small that it ought not to be considered. Therefore, no matter what the nature of the empyema may be, the sole treatment is to open and drain it as soon as possible.

Now and then we meet with a case of this sort: A patient has pneumonia, followed by a pleural effusion, which on exploration is found to be just turbid, but not actually purulent; under such circumstances we may safely wait or be content with drawing off the fluid by paracentesis if it is abundant. There is a chance that pus may not form, but in the great majority of cases it does. In a few days the effusion generally re-accumulates; we explore again and it is found to be definitely purulent, and then it is no use waiting any longer, the pleural cavity should be freely opened and drained.

What shall we do in a case of double empyema? It is met with occasionally, usually after a double pneumonia. The best thing is at once to open and drain one pleural cavity and to draw off the pus from the other side by paracentesis, so avoiding the shock of an immediate double operation. A few days later open and drain the second cavity. There used to be an idea that a patient could not live if both pleural cavities were opened and drained simultaneously, that the lungs would collapse and death result. That, however, is an entire misapprehension, for in these cases the lungs are always fixed by adhesions, so that when the pleural cavity is opened no further collapse occurs. There is, therefore no risk in draining both cavities at once; but it is better, so as to avoid undue shock, to do two separate operations, with an interval of a few days between. Complete recovery may follow unless the empyemas are part of a general septic or pyæmic condition.

If, after an empyema has been drained, the patient does not progress satisfactorily, and especially if there is still some fever, almost invariably there is something wrong with the drainage, or else there is pus in some other part of the body. Sometimes in such cases there is a second loculated empyema which was not opened at the first operation, and when this has been drained all goes well.

It is not for me to speak of those cases in which the lung will not re-expand and in which a large discharging cavity remains. They are exceedingly unsatisfactory, and surgical procedures have been devised to deal with them, involving often, serious and extensive operations—removal, it may be, of large portions of the chest-wall—but with such purely surgical questions I am not concerned.

In conclusion, I would only say that the more we recognise the possibility of overlooking an empyema, especially after an acute pneumonia, the earlier we make the diagnosis and the sooner we freely open and drain the pleural cavity, the less likely shall we be to meet with these troublesome cases, with the necessity for serious operative interference at a subsequent period.

March 29th, 1915.

VARICOCELE IN THE FEMALE.*

By W. E. FOTHERGILL, M.A., B.Sc., M.D.,

Honorary Assistant Gynæcological Surgeon, Manchester Royal Infirmary; Lecturer on Midwifery and Gynæcology, Victoria University.

PROBABLY the commonest of all the complaints made by young women who seek the advice of gynæcologists is that of dull aching pain in the lower part of the abdomen. The pain comes and goes, it is generally on the left side, often on both sides, and sometimes only on the right side. At first it is often associated with menstruation. It may begin before each monthly flow, and may be relieved by the onset of the discharge; or it may continue throughout the period and for two or three days after it is over. Later the pain begins to trouble the patient quite apart from the monthly times. It is made worse by standing, and is generally relieved by lying down. It is often relieved by moderate exercise, but is increased by fatigue. This pain does not afflict young girls, but comes on gradually during early womanhood. Its onset sometimes synchronises with the dawn of sexual feeling. It sometimes, but by no means always, disappears after marriage, indeed it often begins after marriage, and is not relieved by pregnancy and parturition. On the other hand, the pain generally lessens long before reproductive life nears its end, and ceases after the menopause. It is worse when the patient's general health is low, and, in many cases, it disappears during periods of good health, such as holidays. The pain is made worse by constipation.

Physical examination in the majority of cases reveals no abnormality whatever. The vulva, vagina, uterus and appendages are all normal in size, shape, position and consistency. The pain is quite common in virgins and in nulliparous married women. In other words, it is common in patients who have had no infective trouble, either septic or venereal in nature. This pain, in short, is not a result of inflammation, for inflammation does not occur in the female pelvis except as the result of infection.

The pathology of the condition was not known until abdominal sections became common. Recently, however, it has been noted that women who complain of this "pain in the sides" have bunches of dilated and distended veins in the broad and infundibulo-pelvic ligaments. The condition, in fact, is varicocele of the ovarian veins and of the neighbouring veins.

Varicocele is often the cause of pain in the male subject, and it must be realised that it has the same effect in the female.

Until recent years it has been usual to make a diagnosis of *ovaritis* in cases of varicocele in the female. The use of this word implying inflammation has led to the use of treatment appropriate enough for inflammation, but most unsuitable for aching caused by distended veins. It is most desirable that the word ovaritis should be kept for its proper use. It is not the name of a disease, but of a pathological process which occurs as a phase in some cases of pelvic infection. Ovaritis can never be primary, for infection of the ovary must always be secondary to infection elsewhere. Organisms must reach the ovary by way of the uterus and tubes, by way of the peritoneum or by the vessels, and there must be a primary infection in some other part of the body.

If a man has gonorrhœa and the infection reaches the testis, the man has orchitis—a phase of gonorrhœal infection; but it would be quite wrong to say that a man has orchitis when he has only varicocele with aching as a symptom.

Why then should a woman be said to have ovaritis when she has an aching distension of the ovarian veins, but has never had any pelvic infection?

The diagnosis of varicocele in the female must be made by exclusion, for it is not possible to feel the bunches of dilated veins during bimanual examination. If the pain is complained of in the absence of physical signs, and in the absence of any history of pelvic infection either septic or venereal, the case is simple. But this pain may be present together with other pelvic troubles, and when two or more conditions are combined a difficult diagnostic problem may be presented. When the pain is on the right side a common error is the diagnosis of appendicitis. The appendix vermiformis is constantly removed for varicocele in the female. No relief from pain follows the operation, and the removal of the ovary for "ovaritis" often follows with no better result. The

* Clinical Lecture at the Royal Infirmary, Manchester.

patient still complains, and may drift through the hands of a series of quacks and faith-healers into chronic invalidism.

The prognosis is of course good as regards life, health, and reproduction; but the patient with varicocele must be told that she will probably be troubled by her pain whenever she is run down or constipated, until the change of life gives her permanent relief. She is generally much reassured when the nature of the condition is carefully explained. She recognises that it exposes her to no danger or disability, and that she must tolerate the pain, mitigating its severity by attention to her general health, and by keeping her bowels loose. In certain hyperæsthetic young women, very slight distension of the pelvic veins enters consciousness in the form of pain which tends to become permanent or habitual. In these cases the prognosis is bad, but it is the patient's head which is at fault rather than her pelvis—she has "a genius for pain," and will probably become what is known as a neurasthenic.

The treatment of varicocele in the female has in the past been dominated by the diagnosis of ovaritis. The hot douche, the glycerine plug, the blister and the curette have all had their fling, to the detriment of the patient, under the mistaken idea that there was "inflammation" to be dealt with. What would be thought of a medico who put a cantharides blister on a man's scrotum for varicocele? And how would men like to have one or both testes removed because their veins ached? Yet poor women are constantly subjected to treatment on these lines. Even now the square mark of the blister is often seen on the so called ovarian region, and thousands of healthy ovaries have been removed for "pain in the sides." The removal of one ovary never cures a patient. When both are sacrificed the surgical menopause which follows is accompanied, like the natural menopause, by cessation of the pain. But the symptoms of the surgical menopause are often severe and of considerable duration.

Ligature and excision of the bunches of dilated veins has frequently been tried during recent years. It is not easy to do it effectively, as the veins are very numerous and spread over a considerable area. It gives relief from the pain for a time, but after a few months the pain generally returns during some period of temporary ill-health. When some veins have been cut away, no doubt others enlarge and become distended in their turn. Thus there is not as yet any surgical treatment for varicocele in the female.

A correct diagnosis and a good prognosis are the main therapeutic measures available in these cases. The nature of the condition should be explained, and the patient should be assured that the pain does not mean disease, and will have no serious outcome. The use of a saline aperient every morning on waking should be advised, and a mixture containing cascara, strychnine and a little belladonna may be prescibed for use thrice daily when the pain is troublesome. The patient should not stand when she can sit, and should not sit when she can lie down. In the cases of those whose occupation demands constant standing a change of employment may be necessary. Exercise short of fatigue is of great value. Local treatment, such as douching, should be carefully avoided, as it increases the pain by increasing the blood supply of the pelvic organs.

March 29th, 1915.

Injection of Paraffin in Treatment of Ozæna.—Blegvad reports five cured or materially improved over a period of two or three years out of thirty patients whom he has treated by local injections of paraffin. The interval since has been too short in some of the others to estimate the outcome, but the fœtor has disappeared in practically all. The benefit is possibly from the narrowing of the lumen, enabling the nose to be blown more effectually and thus preventing accumulation of crusts, but the modification of conditions in the mucosa is probably the main factor. The mucosa is softened and rendered succulent by preliminary injection of water, so it takes up and holds more of the paraffin. The latter is injected beneath the mucosa on the inferior turbinate and septum. It lifts up the mucosa and then the paraffin is injected directly into the mucosa. There was moderate hæmorrhage in one case and quite severe in another, coming on in the night afterward, but subsiding spontaneously. In two other cases there was brief suppuration in the septum. The ultimate result depends on the regenerating powers of the mucosa. He uses paraffin with a melting point at 51° C.—*Journ. A.M.A.*, lxiv, p. 868.

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We regret that owing to the war the number of pages must be diminished for a time.

POSTURE IN RELATION TO GENERAL ANÆSTHESIA.*

By W. J. McCARDIE, B.A., M.B., B.C.Cantab.,
Anæsthetist to the General, Dental, and Ear and Throat Hospitals, Birmingham.

THE importance of posture in general anæsthesia can hardly be exaggerated. Variation of posture may affect (1) respiration through either the air passages or lungs themselves; (2) it may affect circulation by its influence on the heart's action and also on the blood-vessels; (3) it may affect the nerves, particularly in the limbs; (4) it may affect the muscles and joints in the way of strain, and post-operative aches and weakness; (5) it may affect the internal organs, as, for example, the stomach, intestines or kidneys.

Let us consider the posture of a healthy sleeping child. It lies nearly on its side, and more or less curled up, that is to say, with all its joints (including the vertebral) moderately flexed. This is the natural position, that of greatest ease, and the best for anæsthesia.

With regard to the fixation of large joints during anæsthesia, I would lay it down as a rule that they should never be flexed or extended beyond a right angle. Instances will at once occur to us in which prolonged hyperflexion or hyperextension of certain joints—*e. g.*, the shoulder or the hip—has injured nerves or embarrassed breathing, or both. As a rule, then, all the joints (including the vertebral) should be slightly flexed—*i. e.* in the position of the greatest ease. Another general proposition I would make is that in operations on the upper part of the body, where the nature of the anæsthetic or the needs of the operator do not contra-indicate it, the head and shoulders should be slightly raised in order to lessen oozing and to decrease the amount of the anæsthetic. The need

* Read at the Royal Society of Medicine.

for the escape of mucus will not hinder the raising of the head and shoulders, because its secretion should be stopped by the administration of atropine or scopolamine. On the contrary, in operations on the lower part of the trunk and legs the head and shoulders should be just a trifle lower.

With a view of eliciting discussion, I propose now to make a few remarks on posture during operation on the various regions of the body. Let us begin at the top. In operations on the brain and spinal column the help of elevation may be invoked to lessen hæmorrhage. If the patient is to be laid prone, as for certain brain operations, a separate head-rest, such as that I think used by Harvey Cushing, is advisable, and in order to relieve the respiratory machine as much as possible hard pillows should be placed beneath the clavicles and the iliac crest. Coming now to operations on the mouth or air passages, posture, as a rule, must be subordinated not so much to the lessening of congestion as to the escape of blood from the air passages or the prevention of its entry therein; hence in most cases it is advisable that the head and neck be just so much lowered that the flow of blood will be towards and out of the mouth, and that congestion will only be slight. On the other hand, if methods are to be adopted which absolutely shut off the air passages from the site of operation, it is better to raise the head and neck, as, for example, during the use of Crile's tubes or Hahn's tampon cannula. It is affirmed that during intra-tracheal etherization the entry of blood into the air passages is prevented by the force of the return current around the catheter. This, I presume, holds good during the horizontal or lowered head position of the patient; if it also holds good when the head and shoulders are raised, this method will be the more valuable because it will allow of the advantage of greater anæmia of the parts during the operation. Possibly in this case the pressure at which the etherized air is delivered may be raised a little beyond that usual, in order to increase the force of the return current. I should be glad to hear from those who are skilled in intra-tracheal anæsthesia if they find that the return current is powerful enough to prevent blood entering the larynx when the head and shoulders are well raised or the patient is in the sitting posture. If intra-tracheal anæsthesia be used for operations about the head, neck, or chest, posture as regards mechanical obstruction of the upper air passages need not be considered.

A method of ensuring free breathing which has been much advocated abroad is that of Witzel, who places the head in what he calls forcible dorsal reclination over the end of the table. This method is opposed to the principle that flexion is better than extension, particularly for respiration, and causes strain on the chest. On the other hand, it has the advantage that mucus and foreign matters collect in the naso-pharynx and cannot be inspired, although they may enter the nasal sinuses.

A few words as to anæsthesia when the patient is sitting upright in a dental chair. The principle of flexion applies: the neck should be, if anything, a trifle flexed, and the thighs and knees should be flexed to a right angle. To help to secure flexion, and to prevent movement and purchase for muscular spasm in the chair, we have had made at the Dental Hospital in Birmingham a special chair, in which the seat is at less than a right angle to the back, and the foot-board is set at an angle of about 75° to the leg part of the chair. The back, seat and the footboard are covered with corrugated rubber, which grips the patient's clothes and feet somewhat, and tends to prevent slipping and extension of the body and limbs. Instead of the ordinary footpiece there is merely a flat board at an angle of about 15° with the horizontal line, so that if the patient's feet shoot out they cannot be caught in any projection or hollow, and therefore get no purchase. I prefer the dental chair for operations on the Gasserian ganglion, and keep the patient sitting bolt upright.

With regard to anæsthesia in the sitting posture, Chaldecott says that he has administered chloroform for six years to patients of all types and ages sitting quite upright, and his opinion is that in nose and throat operations under chloroform the upright position is the safer, and the alleged danger of syncope does not occur, even in subjects of chronic asthma, cardiac dilatation, or goitre, because breathing is easier when sitting up than lying down. We must remember, however, that he induces anæsthesia rapidly with gas and ether, and that his operations apparently were only those that needed a light anæsthesia. Personally, I very often give chloroform as he does after induction by ethyl chloride and ether for prolonged dental operations. By the way, I should like here to join

with Sir Frederic Hewitt in his condemnation of the semi-recumbent posture for dental or other operations. It is the worst one possible.

In operations on the upper part of the body, especially those in which there is likely to be free bleeding because of the vascularity of the parts or extent of the wound, I think it is wise, if ether is being given, slightly to raise the head and shoulders. This will lessen hæmorrhage and congestion, and even from the commencement of induction, according to Hornabrook, less ether is needed. Besides, the anæsthetist has something to fall back upon should danger arise, because he can flood the vital parts with blood by lowering the head and shoulders.

In operations on the breast the arm should never, or for as short a time as possible, be extended beyond a right angle, particularly in stiffly built, fat people, because of the danger of brachial paralysis.

In operations on the lung and pleura we have to remember that the sound lung must lie as free as possible and that its bronchus should be above the level of the bronchus of the unhealthy organ, so that no infection of the sound one can take place. To help in this some slight degree of the raised pelvic position should be utilised.

A few words as to the movements of the diaphragm as shown by radiography. During ordinary normal breathing the diaphragm moves only about $\frac{1}{2}$ in., but with forced respiration it moves $2\frac{3}{4}$ in. on the left side and $2\frac{1}{2}$ in. on the right side. Hence any marked hindrance to respiration involves much strain on the lungs.

Trendelenburg Position.

Articles have been written on and many instances given of the dangers of the Trendelenburg posture. It gives much advantage to the surgeon and has many disadvantages from the patient's and anæsthetist's points of view. As has been said, anæsthetics may have been unjustly blamed for complications really induced by elevation of the pelvis. It is advisable that the inclination of the body should not exceed an angle of 45°. Here I would like to quote on the general question some remarks by Kocher. He very pertinently says: "A man cannot be expected to stand on his head for over half an hour without his circulation being seriously affected, and this is obvious to anyone who has a knowledge of the mechanics of the circulation and who has witnessed the copious venous hæmorrhage associated with a dependent position of the head as in Rose's operation. Obese people and those who suffer from disease of heart or vessels can often obtain sleep without respiratory embarrassment only when the head and shoulders are raised." How, then, can they safely bear the Trendelenburg posture?

Franz, who investigated the behaviour of respiration and circulation in the Trendelenburg position, found that abdominal respiration without anæsthesia was constantly weakened and thoracic breathing was little, if at all, compensatorily strengthened. During chloroform narcosis there was rather greater compensation for the weakened abdominal breathing by deepened thoracic respiration. On the whole the raised pelvic position lowered the ventilation of the lung in no small number of cases. If patients are suddenly raised to the normal position there is, with the first breath, marked lessening of abdominal and thoracic breathing, hence danger of collapse from lessened elimination. Franz asserted that there was no special damage to patients under chloroform through the Trendelenburg position and that there was very little increase in the percentage of bronchitis. He found that in the case of ether there was considerably more bronchitis after operations in the Trendelenburg than in the horizontal position.

Dr. Thomas Wilson, Gynæcologist to the General Hospital, Birmingham, some years ago, collected statistics of the relative frequency of pneumonia after operations in the horizontal and Trendelenburg postures. In 144 cases operated upon in the horizontal posture he had seven pneumonias; after the Trendelenburg there were ten pneumonias. These figures relate to a time previous to the use of scopolamine and morphine.

Franz noted in 825 cases of ether narcosis in the dorsal posture nineteen (2·3 per cent.) cases of bronchitis, while in 493 in which pelvis was raised there were forty-four cases (8·9 per cent.); 150 chloroform narcoses with the patient lying dorsally were followed by four (2·7 per cent.) cases of bronchitis and in 233 cases in the Trendelenburg position by nine (3·9 per cent.), showing that with chloroform the difference is much less marked than with ether. Franz explains that frequency of bronchitis after operations in the raised pelvic

posture is due to the escape of infective mouth contents into the bronchi after the patient is raised, and suggests the advisability of carefully cleansing the mouth before the change is slowly made to the dorsal position.

To illustrate the influence of the Trendelenburg posture in the causation of pulmonary embolism I will quote Zweifel, who found that out of 1800 operations there were eighteen deaths from pulmonary embolism. Afterwards he no longer kept the patient's legs flexed and firmly bandaged, but placed them loosely in horizontal frames. The shoulders were supported, and the Trendelenburg position was adopted as seldom as possible. After this change the deaths from pulmonary embolism were reduced to three in 800 operations. Zweifel considers that the position favours thrombosis both by facilitating sepsis and also by its mechanical action on the blood-vessels.* Intestinal obstruction, surgical emphysema, apoplexy, and also acute post-operative dilatation of the stomach have been caused by this posture.

Bovée studied eight cases of ether anæsthesia and eight of chloroform anæsthesia and found that "almost no urine is received in the bladder while the Trendelenburg posture is being employed. If it can be concluded that the renal function is greatly lessened while the patient is in the Trendelenburg posture then the dangers of that posture are at once appreciated. In renal inefficiency it would seem that the use of the Trendelenburg posture would introduce a special element of danger, and this would be more marked when ether was used than when chloroform was employed as the anæsthetic."†

Hewitt records a case in which aphasia and renal hemiplegia followed operation in this position; the patient was twelve months in recovering. Kirkby Thomas noted a case in which the patient was quite blind for three days and had delusions for a week.

Gatch, Gann, and Mann‡ said that a gynæcologist of wide experience once informed them that he knew of no more certain way of killing a fat woman than by leaving her for some time in the high Trendelenburg position, and they record the sudden death of a patient with aortic and mitral insufficiency and pericardial effusion who had been anæsthetized with gas and oxygen and ether in the flat position and was then placed in the lithotomy position with pelvis well elevated. They etherized dogs without any morphine and put them in the Trendelenburg position at 45° to 50° of inclination and took records of respiration and blood-pressure. The dogs died on an average in twenty minutes. The records showed that the change to head-down position caused a slight rise in blood-pressure, a slight increase in the pulse-rate, and little or no change in the rate of respiration. The breathing, however, became altered and assumed a diaphragmatic character. Respiration failed first and then pulse and blood-pressure, which hitherto had practically shown no change, also failed. It was at first thought that death was brought about by fatigue of the diaphragm, but they found that a dog under morphine narcosis will live for hours in the head-down position without any sign of fatigue of the diaphragm; a fact demonstrated by Hill and verified by them. They conclude that failure of respiration is in some way caused by the ether. They found that the ill-effects of the head-down position are due primarily to failure of respiration and were prevented if respiration were kept stimulated by hypercapnia. Gatch, Gann, and Mann also found that in dogs the heart failed much more quickly from asphyxia in the head-down than in the horizontal position. They conclude that "in normal men or animals under anæsthesia the head-down position has no ill-effects provided that the respiration is adequate and the muscles are at rest. When the breathing becomes insufficient, the entire mechanism wherewith the body compensates for the effects of gravity on the circulation is deranged. The asphyxia injures the cardiac muscle and raises the blood-pressure. The head-down position at the same time insures that every drop of blood which passes through the capillaries of the abdomen and legs will quickly return to the heart, a condition not present when asphyxia occurs with the body in the horizontal or upright position. The heart then has to pump a supply of blood at least normal against an increased arterial pressure, at a time when it is poorly able to respond to the increased strain. If the patient at this time struggles or if pressure is made on his intestines the state of affairs is rendered much worse."

* 'Brit. Med. Journ.', 1911, ii (Epitome), p. 19.

† 'Journ. Amer. Med. Assoc.,' Chicago, 1910, liv, p. 1994.

‡ 'Ibid.,' 1913, lx, p. 1273.

The conclusion of the problem of the Trendelenburg position is that it should generally be used for as short a time as possible and not used at all in patients suffering from obesity, obstructed breathing, cardiac, renal, or arterial disease. The legs should not hang, but be supported as mentioned a little later, in order to prevent tension on the abdominal muscles and also the danger of thrombosis. The shoulders should be fixed to support the body.

With regard to posture in abdominal operations, generally the principle of flexion applies, as by it muscular relaxation is facilitated, strain on the back and joints is prevented, and post-operative pain and discomfort are done away with. Goldthwait has aptly pointed out that the assumption of a faulty position without support for the back and with the thighs slightly extended as in the ordinary Trendelenburg position has resulted in strain of the lumbar spine and sacro-iliac joints. He states that only those who have to do with patients suffering from weak or painful back or with chronic joint diseases can appreciate how frequently conditions of strain arise. If a patient be in the Trendelenburg position, the legs should be kept straight, a pillow should be placed under the lumbar spine to support it and also under the thighs to prevent hyper-extension, in fact, to flex them slightly on the abdomen; thus no strain of the lumbar or sacro-iliac joints can result.* He writes that "if the bed or supporting structure is hard, so that the buttocks cannot settle down into it, the lumbar spine must sag downwards, and if the buttocks are large or the waist small the amount of this sag may be enough to cause painful strain on the interspinous ligaments or on the sacro-iliac joints." The result is that the strained sacro-iliac joints cause distressing backaches and possible after-disability.

These remarks apply also to the horizontal position. During gall-bladder operations, when a large pillow is placed under the liver, the patient's head should be well raised and a thick pillow placed under the thighs and legs. By this means the pull of the head and neck and of the legs will be taken from the abdominal muscles, longitudinal tension will be greatly lessened, lateral relaxation will be increased, and total relaxation of abdominal muscles and fasciæ will be much more easily obtained. I believe that post-operative backache in many cases, more particularly in muscular men, will be prevented by adopting Goldthwait's suggestion.

With regard to the placing of pillows under the liver and kidneys, it is advisable that they should always be soft to prevent bruising and to allow of some movement of the ribs during respiration. A great deal of the rigidity often complained of by surgeons during abdominal operations is, I am sure, due not only to some slight degree of asphyxia but also to the mechanical strain of muscles and fasciæ, which strain can be largely prevented by a properly arranged posture.

In the lithotomy position the head, for reasons already mentioned, should be well raised, a soft pillow placed under the small of the back, and in no case should the thighs be bent towards the abdomen at more than a right angle; otherwise interference with abdominal respiration will occur.

Post-operative posture should be the natural sleeping one mentioned at the beginning of my remarks, that is to say, the patient should be laid well on the side with all the joints flexed. When he comes round, if vomiting persists the sitting-up posture will often stop it. Persistent after-vomiting may occasionally be due to some degree of dilatation of the stomach. If the patient be supine the stomach may not be able to empty itself, and the sitting-up position will allow of it doing this, and also allow of better lung ventilation. Occasionally we see that dreadful condition called acute dilatation of the stomach, the symptoms of which are vomiting, abdominal distension, collapse, epigastric pain, and thirst. The cause of this condition is usually mechanical obstruction contributed to by a recumbent posture. The usual treatment is the use of the stomach-tube, but Borchgrevink, of Christiania,* advocates as a cure, which is often at once brilliantly successful, the adoption of the prone posture. The patient who has been almost *in extremis* suddenly recovers. The prone posture was first suggested, he says, by Schnitzler, and has been recommended by Moynihan. The explanation of the success of this posture is as follows: The stomach does not suffer

* 'Journ. Amer. Med. Assoc.,' Chicago, 1911, lvi, p. 642.

* 'Surg. Gyn. and Obstet.,' 1913, xvi, p. 662.

more than any other organ from the effect of the general anæsthetic, debility, etc., but it is the one organ on which demands are made almost at once after a major operation, and if the demands are beyond its functional capacity at the moment, the stomach simply abandons the attempt to respond to them. Therefore we must not make demands on the organ until it has regained its normal tone after operations, and we must refrain from overloading it. The effect of change of posture from the recumbent to the sitting in increasing lung ventilation and making easier the expectoration is well known.

April 5th, 1915.

An Unusual Case of Poisoning by Cantharides.—Dandy reports the case of a man, æt. 60 years, suffering from stricture of the urethra and albuminuria, who took two pills, each containing 5 cgm. of powdered cantharides, apparently for its aphrodisiac effects. Severe dysuria began twelve hours afterwards, urine being passed frequently but in very small quantities and mixed with blood. There were an intolerable burning sensation in the hypogastrium, acute enteritis, and blood-stained stools. When seen by the writer forty-eight hours after taking the pills he presented the symptoms of intense pulmonary congestion, and could speak with difficulty. During the previous ten hours he could pass urine only drop by drop, and a gradually increasing dyspnœa had set in which he regarded as a severe attack of asthma. His condition was due to a very acute œdema of the lungs depending on nephritis, although he had never previously presented pulmonary symptoms. In spite of free venesection the pulmonary œdema increased, the dyspnœa becoming rapidly more severe. The skin, at first pale, became cyanosed and of a deep purple colour, while the nails were almost black. The pulse became uncountable with chilling of the extremities, and the patient presented great anxiety and terror, but remained conscious to the end. He died about fifty-one hours after swallowing the drug. The writer directs attention to the danger of cantharides, especially in cases of nephritis and the unusual symptoms it may produce in such cases.—*Journ. de Méd. et de Chirurgie Prat.*

APPENDICITIS IN THE ELDERLY.*

By G. P. NEWBOLT, F.R.C.S.Eng.,
Examiner and Lecturer in Clinical Surgery, University of Liverpool; Surgeon, Royal Southern Hospital, Liverpool.

THE diagnosis of appendicitis is not always as simple as is generally supposed, and I wish to draw your attention to one or two cases occurring in elderly people. By elderly people I mean those whose ages range from sixty years upwards.

In the year 1902 I saw an old gentleman, in consultation, who had been ill for some time, no correct diagnosis having been made, although he had been seen many times in consultation. As far as I remember, he had occasional rigors, with attacks of dyspepsia, and a big liver. Eventually a swelling formed at his external abdominal ring, and this spread into his scrotum. It was at this time that I saw him. I opened the swelling under localised anæsthesia, and some foul pus escaped. One or two incisions were made later on, but he gradually got weaker, and eventually died. He had an appendicitis with cellulitis spreading up behind the cæcum and down towards and into the cord on the right side. As far as I recollect, there was no tenderness in his right iliac fossa when I first saw him, and I have never seen a similar case. Apparently it was a slowly spreading cellulitis due to a perforated appendix with escape of septic fluid into the cellular tissues. The patient was of eccentric disposition. The point, however, to which I wish to draw attention is the fact that you may get a perforated appendix not giving rise to very acute symptoms at the time, which may cause an abdominal condition most difficult to diagnose. Again, as the condition is very serious, a correct diagnosis is of great value.

These cases are not common. I have notes of three out of some four or five hundred cases operated upon. Most surgeons are aware of the cases labelled intestinal obstruction which are really cases of general peritonitis following appendicitis, and even though the symptoms are

* Read at the Liverpool Medical Institute.

disguised by morphia it is generally possible to arrive at a diagnosis.

In my experience most cases of appendicitis occurring in elderly people run the same course as those occurring in others. In many of the cases abscess has occurred, probably from the fact that operation has been postponed as long as possible because they were elderly.

The first case to which I wish to draw your attention occurred in a woman, æt. 60 years, who was sent in to the Southern Hospital on 13th October, 1913, with a diagnosis of intestinal obstruction. She had been ill for three days, and was greatly distended. Her pulse was 110, she was vomiting; no flatus was passing, but her bowels had acted slightly twenty-four hours before admission. Nothing abnormal could be felt per rectum, there was no history of any gynæcological trouble, no gall-stone history and no hernia. Her temperature was 98·4, and there was no tenderness in the right iliac fossa and no muscular rigidity.

I made a median incision low down, and found purulent fluid in her pelvis. Her ovaries were normal. On feeling in the appendix region, I discovered a concretion which at once cleared up the diagnosis. The appendix was cut down upon and removed—a sloughy hole existing in its base. She made a slow but good recovery; the distension existed for a fortnight in spite of pituitary and enemata, even though the bowels acted. The median wound gaped. The woman was in poor health, with thin abdominal walls, and evidently of the type in which the tissues are lax and the inflammatory process subacute.

In the second case the symptoms were just as misleading as in the first, or more so.

The patient was a male, æt. 68 years, who had suffered from digestive troubles for some months.

His bowels had acted well on a Tuesday. He had vomited that day, but had no abdominal tenderness. His pulse and temperature were normal. He had had some pain in his epigastrium, but nothing more than he was accustomed to have. Two days later, on the Thursday, he started fæcal vomiting; no flatus had passed for two days in spite of enemata: there was no hernia, no tenderness over the abdomen, but he was distended and seemed to be suffering from intestinal obstruction. His pulse was 80 and his temperature normal. I was asked to operate upon him for intestinal obstruction. A median incision was made, and on opening the abdomen I found the omentum fixed down in the pelvis and the pelvis filled with stinking fæcal fluid of the worst kind. There was a perforation in the base of his appendix. I removed the appendix and drained him above the pubis and in the right iliac region. He lived about nine days after operation, but foul, offensive fluid was discharged, and eventually the suprapubic wound became gangrenous. He vomited and hiccoughed incessantly, and died, evidently poisoned by the septic contents of his abdomen. His pulse kept at 80–84, and his temperature was subnormal until twenty-four hours before his death. His bowels acted freely and his distension greatly subsided. He was not a vigorous type of man, had probably lived freely, and was in poor health at the time of his attack; in other words, he had no recuperative powers with which to deal with his infection.

I think these cases are very interesting and instructive. Had either of them complained of pain in the appendix region, a diagnosis might have been made. They were both distended and both were vomiting; in neither was there any temperature. In the male there was a clear history of loss of weight, and I thought I should find a malignant stricture of the large intestine. There was no peristalsis in either, though the distended coils of intestine could be seen in the female case.

Can these be diagnosed? I do not think they always can, but there are points which may throw a good deal of light on them.

Firstly, the knowledge that this type of case exists, and that with a pulse of 80 you may have acute disease. The presence of a high leucocytosis points to a peritoneal infection as against mechanical obstruction.

The fact that in mechanical obstruction the passage of flatus is usually stopped absolutely, though this is not always the case in peritonitis.

Having excluded the common causes of obstruction, and being in doubt, it is always well to keep the appendix in mind.

Re-examination under the anæsthetic prior to operation is always valuable. Pulse and temperature often give no guidance whatever. A rise of temperature helps, but its absence means nothing. In mechanical obstruction there is usually peristalsis.

Pain at the onset of the illness is as often due to the nipping of the bowel as to perforation.

Fæcal vomiting may be present in either peritoneal or mechanical obstruction. The treatment is surgical, and when in doubt it is a good plan to make a small incision low down above the pubis in which a tube may be placed later on. If there is fluid, the nature of it is often diagnostic, though not always so. If fæcal, the appendix should be exposed through an incision over it, and removal and drainage carried out. If the abdomen is clean and the fluid clear, the median incision can be enlarged upwards and the obstruction sought for. In the inflammatory cases there may be a little trouble in opening the abdomen due to omental adhesion. In conclusion, I think you will all agree that the diagnosis of a case in which the abdomen is distended after four or five days' illness presents countless difficulties, and the object of my note is to draw attention to some of them.

April 5th, 1915.

MENTALLY DEFECTIVE CHILDREN. By ALFRED BINET and TH. SIMON, M.D. Authorised Translation by W. B. DRUMMOND, M.B., F.R.C.P.(Edin.). London: Edwin Arnold. Price *2s. 6d.* net.

The authors have succeeded in compressing a large amount of information into small space at a remarkably cheap cost. Recent legislation and the Board of Control have forced the pace, both in regard to the definition and the limitation of the term mentally defective. The statute says that the examiners shall be three in number, viz., the head of a special school, an elementary school inspector, and a doctor.

The precise *rôle* of the doctor is interesting. "It seems to us, in fact, that there is often some confusion as to his attributes, and two opposite tendencies may be recognised. According to one—the more widespread—the defective are often, if not always, invalids, and belong to him by right. It would be an encroachment on his privileges to concern oneself with them. The opposite opinion consists in not committing to him any particular authority in the matter. This is the case in Germany, where there are schoolmasters who carry things with a high hand in the special schools. Let us add that the doctors themselves have done nothing to bring about an *entente*. Speak of defectives before them and they say, 'That is our business,' and they are perfectly right; but having affirmed their right they pay very little attention to the territory they defend."

In an appendix will be found the Binet-Simon Tests of Intelligence. We commend this book on a subject, the exact sphere of which the medical man will soon be called upon to decide.

SQUIRE'S POCKET COMPANION TO THE BRITISH PHARMACOPŒIA, 1914. Second Edition (16mo). Pp. 1040. London: J. & A. Churchill. Price *10s. 6d.*

The first edition of this pocket companion was published in 1904, and originated in the necessity for dividing the older 'Companion to the British Pharmacopœia' into two publications. This was the result of the very great increase in the literature dealing with modern scientific research which has necessarily to be reviewed in the work. The present is the second edition and follows the style now made classical by the 'Companion.' Under solubility, dose, prescribing notes and incompatibles, is a mine of useful and practical information for the medical man. The prescribing notes which will most appeal to the latter have been systematically revised and considerably enlarged.

A chapter on "Therapeutic Agents of Microbial Origin" has been revised and rewritten by Prof. Hewlett, of London University. This includes up-to-date information on antitoxins, serums, tuberculins, vaccines, etc. There is also a chapter on the "Spas of Europe," giving locality, mean temperature, season, etc. The prescriber's convenience is studied right into the index, a clear distinction being there made between official and non-official preparations. The book is a compact little volume and supplies the prescriber with a systematic and complete account of all the additions and alterations of the new British Pharmacopœia. We bespeak for the book a cordial reception.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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NOTICE.

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We regret that owing to the war the number of pages must be diminished for a time.

ON A CASE OF CONGENITAL DISEASE OF THE HEART, PRESUMABLY CONSTRICTION WITH ARRESTED DEVELOPMENT OF THE INFUNDIBULUM (BULBUS CORDIS) AND PULMONARY ARTERY AND PATENT SEPTUM VENTRICULORUM.*

By Sir RICHARD DOUGLAS POWELL, Bart., K.C.V.O., M.D.,

Consulting Physician and Emeritus Lecturer on Medicine at the Middlesex Hospital.

MR. —, æt. 20 years, was sent up to me from the country in February, 1914, by Dr. Young of Leicester, for an opinion with regard to his heart. He was an auctioneer's clerk engaged in light work in his father's office. He complained of hot perspirations coming on several times in the day on the slightest exertion, or on entering warm rooms, but not troubling him at night. These perspirations were so troublesome that he had to change his socks two or three times a day. A walk of 200 yards would bring them on; they caused him to feel oppressed but not faint. These flushings and sweatings came on a fortnight after a third attack of influenza in September of the preceding year. He was able to keep about and do his light secretarial work, and I was especially consulted with regard to what he might do in the way of exercises in view of the condition of the heart.

I found that he had suffered since early infancy from some cardiac defect, and was informed by Dr. Young, who had known him since the age of 4 years, that he had at that time recognised a condition of heart which he regarded as congenital. The doctor who attended at the patient's birth had also observed the heart to be wrong; there had, however, never been any cyanosis. The signs at the age of 4 years were relatively slight and had gradually increased, but up to October,

* Clinical Lecture at the Middlesex Hospital.

1913, there were no notable symptoms. The influenzal attack in September had been slight, with very moderate rise of temperature for two or three days only. The lungs were, however, congested out of proportion to the general symptoms, there being some blood staining of the sputum, and abundant crepitations over the bases. In January again he had a "feverish cold." He had never had any symptoms pointing to rheumatic fever nor any other serious illnesses.

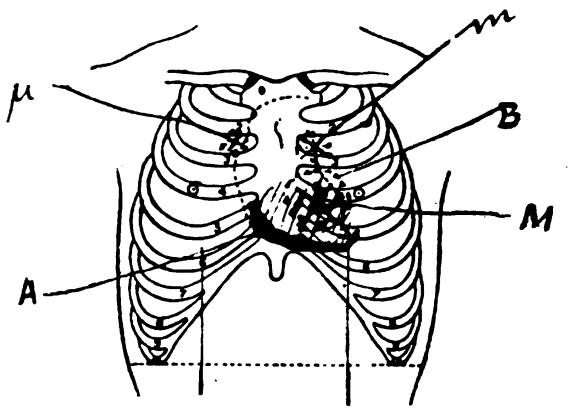
The patient was a fairly well-developed man, rather below the medium build, but presenting no external signs of defective development. There was no notable cyanosis and no clubbing of the finger ends. The pulse was somewhat deficient in power, but presented no other notable characters.

The points to be observed about his heart were that the apex beat was in the nipple line, the impulse of the right ventricle labouring, hypertrophic. There was a systolic thrill over the area marked B, where a loud, rasping, systolic bruit was heard, having its centre of intensity at the fourth cartilage, M, left sternum and well conducted up the sternum and to left, *m*. The murmur was distinctly but less loudly heard, less rough and of higher pitch on the right side, μ. It was rapidly lost as the stethoscope passed beyond the left confines of the heart, and was similarly lost (especially when the patient was reclining) below the margin of the right ventricle at the base of the ensiform. The murmur was audible over the first and second dorsal spines posteriorly. There was no enlargement of the liver, no albumen in the urine; the lungs were clear and the other organs healthy.

It is clear that in this case we have some obstruction to the escape of blood from the right ventricle. The hypertrophy of the right ventricle and the position, time and direction of the murmur point to this.

The situation and direction of the murmur in association with the systolic thrill about the pulmonary area might suggest this obstruction as due either to a narrowing of the pulmonary artery with partial welding of the margins of its valves, or a constriction of the whole infundibulum with imperfection of the upper part of the septum ventriculorum, and perhaps patent ductus arteriosus.

I would have you note, however, that the point of intensity of the murmur is low for pulmonary



A. Hypertrophied right ventricle. B. Dotted outline of area of systolic thrill. M. Point of greatest intensity of rough systolic murmur. *m*. Best conducted with low pitch. μ. Less loudly conducted and of higher pitch. The murmur was also audible at the second and third dorsal spines.

stenosis, and especially for that form of pulmonary stenosis with partially welded valves, in which a thrill would be present.

I have little doubt that the obstruction which produces the murmur and the faulty heart function in this case is at the lower end of the infundibulum, at that portion at which the infundibulum normally merges with the rest of the right ventricle, a point which is only slightly indicated in the healthy human heart, but which is well marked in fœtal hearts up to the fourth week, and in the hearts of the lower vertebrata.

Let me here diverge a little to remind you of what you probably already know of a developmental change of the heart, the pathological bearing of which has, however, only been demonstrated within the last few years, although, I think the classical work of Dr. Peacock shows that he was not altogether unaware of it.*

The tendency just now is rather to minimise the influence of heredity in disease, although we are every day becoming more aware of the abiding effects of our ancestry upon our bodily construction and functional conditions both in health and disease. Last year in discussing the innervation of the heart with special reference to the action of digitalis I alluded to the rhythm of its action being largely due to remnants of fœtal or ancestral structure still remaining in certain portions. Certainly many congenital defects of the heart are

* 'Malformations of the Heart.' T. B. Peacock, M.D. Second edition, p. 32.

due to local developmental arrests rather than to disease. This is true also with regard to other organs, and in taking up the study of the diseases of any particular organ I would advise you to look back to ancestral anatomy and to the epitome of it to be found in fœtal development.

Constriction of the passage from the right ventricle into the pulmonary artery may take place either in the vessel itself or its valves, which may become partially welded together, or at either the pulmonary or ventricular end of the infundibulum or bulbus cordis. And it is common for either of these conditions to be associated with—indeed to be a part of—an atrophy or arrested development of the whole infundibular portion of the heart, which, as you know, is the upper part of the right ventricle, originally forming the last of the four pulsating chambers of the primitive vertebrate heart (Keith).

In some fishes the bulbus cordis is supplied with valves both at the hither and the distal ends, its upper and lower orifices. In the course of the evolutionary changes which are mainly completed in the fourth week of intra-uterine life, the sinus venosus becomes merged in the auricle and the bulbus incorporated with the ventricle, and, as the ventricle becomes divided by a septum proceeding from below upwards, this bulbus portion becomes shut off from the systemic side of the circulation and restricted to the right side of the heart, thus forming an essential part of the respiratory system of the circulation. Professor Keith * has little doubt that the function of the bulbus in the lower vertebrata is chiefly connected with the regulation of the pulmonary circulation, and it is not surprising, therefore, that with the development of specialised air-breathing lungs this portion of the heart should be told off to the pulmonary side. In the normal heart an indication of the original division can still be seen at the point of junction of the infundibular portion with the ventricle proper, and in cases of congenital defect this line of division may be marked to any degree, from a barely notable ridge to an almost complete diaphragm. Thus we may have varying degrees of narrowing at this end of the bulb, as well as at its junction with the pulmonary artery. In combination with narrowing of the lower end of the infundibulum there is often to be observed more or less atrophy of the whole bulbus cordis, including a narrowing of the pulmonary artery with defective or adherent valves. And this deformity of the pulmonary arterial end is frequently the outstanding defect present.

That in most cases of atresia, whether it be manifested especially at one or other end of the infundibulum, the defect is due not to fœtal endocarditis but to changes consequent upon arrested development of the whole infundibular or bulbus portion is the conclusion arrived at by Professor Keith after an examination of a large number of specimens, and in the light of the evolutionary changes effected in the heart which are epitomised in the heart of the fœtus especially before the fourth week, and have been carefully studied by His and by Greil of Innsbruck (1903) and himself.

Dr. Peacock, whose classical work (second edition, 1866) includes all that could be said of congenital cardiac defects up to and far beyond his time, was not unaware of this origin of pulmonary constriction. He observes that the source of obstruction may be (1) at the outlet of the pulmonary artery or its valves, (2) at the upper termination of the infundibular portion of the ventricle, (3) in the course of the pulmonary artery, or (4) at the commencement of the infundibular portion of the right ventricle. It would seem to be nearer the truth to say that in the majority of cases all these conditions are more or less combined as the result of arrested development. In the case now under consideration I believe, for reasons I have given, that the special feature is a narrowing at the ventricular end of the bulb.

In the majority of cases of constricted infundibulum, the septum of the ventricles is deficient, so that there is an orifice, large or small, which permits the blood to escape from the right ventricle into the left and so to the aorta. In extreme cases of atresia of the pulmonary orifice the ductus arteriosus may remain freely patent so as to permit of the supply of blood to the pulmonary artery through the aorta.

It is a question whether some of the cases of so-called hæmic murmurs at the base of the heart are not really cases of slight congenital narrowing at the junction of the bulb with the ventricle. These murmurs are often very persistent when other

* "Malformation of the Bulbus Cordis; an unrecognised Division of the Human Heart," Arthur Keith, M.D. 'Quartercentenary Studies in Pathology,' edited by W. Bulloch, M.D., Aberdeen, 1906, p. 59.

signs of anæmia are wanting, and, indeed, in my experience, the best cardiac evidences of anæmia are often to be found in a temporary reflux through the mitral valve.

It is to be observed that in fishes the contraction wave in the fourth cardiac cavity (the bulbus) follows upon that of the ventricle proper. In most of the serious cases of bulbo-pulmonary narrowing the infundibular portion of the ventricle is probably out of action, forming little more than a part of the arterial tube. There are cases, on the other hand, of slight constriction at the sinus end which may possibly account for a reduplication of the first sound with slight "presystolic" murmur which one occasionally hears. I met with an instance a few days ago which might possibly be so interpreted in a lady who had been credited with "a weak heart" all her life.

I am sorry I have not been able to get the subject of this lecture up for an electric tracing of the heart's action, which might or might not throw further light upon the case, and be useful for reference in other cases; especially those which might be confounded with cases of mitral stenosis.

I have here through the courtesy of the Curator of the Museum of the Royal College of Surgeons a series of six specimens illustrating degrees of this defect of the bulbus and pulmonary artery.

The conclusion respecting the case before us is, then, that there is annular constriction at the junction of the infundibulum or bulbus with the rest of the ventricle or sinus, and that this bottle-neck constriction is almost certainly associated with two other developmental defects which usually accompany it, viz., some narrowing of the pulmonary artery and an incomplete septum ventriculorum. The latter defect may be regarded as compensatory, permitting the obstructed ventricle partially to empty itself into the left ventricle. It varies greatly in degree, and in some cases, as in a specimen presented to the college by Dr. Hay of Liverpool, it is not present. Where the obstruction is extreme, the ductus arteriosus remains patent, so that the pulmonary artery can be supplied through it from the aorta.

Now, what is the prognosis in these cases? If the constriction be very extreme at either the upper or lower orifice of the bulbus, and it may be in either case so extreme as only to permit of the passage of a probe, and in one case in the Museum of the College of Surgeons, presented by Dr. Basil Price in 1911, the whole infundibulum was obliterated; under these conditions life cannot long be sustained—a few days, weeks or months. The minor degrees of constriction at either point are compatible with a considerable duration of life. Dr. Peacock quotes one case in which the patient, a woman, lived to 56 years of age.* Our patient is 20 and may, I hope, live for many years yet. As I have pointed out, there may be cases which have generally escaped notice, in which the narrowing is very slight at either orifice, and the patients are at very little inconvenience, perhaps only incapable of great exertion, and easily breathless; they are regarded as anæmic, and a soft systolic bruit over the pulmonary area is held to confirm this view. They die from other diseases, and the cardiac defect may be altogether overlooked.

In estimating the prognosis of chronic disease, you have, as it were, to look behind the lesion—how far is it compensated and in what degree are the reserves called up? In kidney disease, for instance, there are certain symptoms; you find albumen in the urine and casts of sorts, by which you estimate the kind of lesion present, but you must look at the output of urine, its specific gravity, and the amount of urea excreted in the twenty-four hours in order to estimate what reserve of power there is.

In cardiac affections the problem is a similar one; you estimate the force exercised behind the lesion, the output at a peripheral vessel, *e.g.* the pulse, and the regularity or disorder with which the heart is working (*a*) under conditions of rest, (*b*) under varied degrees of exertion. The cardiac and respiratory functions are so closely associated that an obvious symptom of the patient, distress of breathing under degrees of exercise, is of considerable value as a guide. In our case we find a labouring hypertrophied right ventricle acting with regularity and force to compensate for the obstruction by its increased diligence.

In the history, however, we note that a mild attack of influenza with very little pyrexia was associated with marked congestion of the lungs and some exudation of blood with the sputum. Since that time the youth has felt his heart weakness, of which he personally had not been conscious

* *Loc. cit.*, p. 88.

before. We may assume that the circulation through the lungs is maintained with bare efficiency by a hard working right heart, and that there is but little reserve power in this direction.

There is another source of danger under which these congenital heart cases tend to terminate, viz. by the occurrence of endocarditis, either simple or infective, at the seat of constriction. This is one of the most common ways in which such congenital defects start on a fresh and downward course, and not a few of them have terminated by an ulcerative or infective endocarditis, such as you see illustrated in this heart contributed by Dr. Hay to the College of Surgeons Museum. The specimen is from a woman aged twenty. The right ventricle is subdivided by a perforate septum. The ventricle is greatly hypertrophied; the infundibulum reduced, and its lower orifice bounded by a fibrous margin. The septum ventriculorum is not perforated. The obstruction and the wall of the infundibulum are the seat of ulcerative endocarditis.

You know how common it is for an infective endocarditis to occur at the seat of an old endocardial valve lesion, presumably because tissues which are the seat of old inflamed lesions are less vitalised and less protected by endothelium, and further in consequence of the general law that endocardial and endoarterial lesions are apt to occur at positions of strain. On both these grounds an obstruction, the result of imperfect development, would be less protected and more liable to endocarditis of an ordinary or an infective origin.

This secondary appearance of endocarditis has been regarded as indicating that the primary cause of the deformity or defective development has been a fœtal endocarditis. There is no sufficient evidence that this is the case.

Treatment.—You cannot alter the heart condition. You can, however, control the life within its limitations. You must not deprive the heart altogether of the stimulus of exertion, or it will get lazy and atonic. You must not put too much stress upon it, or its slight reserve powers will be overtaxed. You must not check your patient at every turn and fidget him with restrictions or you will make life not worth living and so lessen his will power.

To put these views in a practical form I will quote the latter portion of my letter of advice about him. "As regards treatment:

"(1) The patient must have regard to his limitations, *i. e.* not to overtax an always labouring right ventricle by straining or fatiguing exercises, but at the same time not to be an invalid misery. I therefore told him that at cricket he might do batting and wicket-keeping, but he must not scout or bowl or run. He might play moderate golf or do some covert shooting or motor driving (but must not start the machine). An occasional twenty-four hours in bed on any excuse, and perhaps in any case once a week or fortnight, will do him good, but to lay him by would only enervate the heart and, I think, precipitate the trouble. I advised him to continue his morning's work and to have a rest after lunch.

"(2) I look upon his sweatings as arising from nervous depression after influenza and would try:

℞ Liq strychninæ, ♏xxxvi.
Acidi phosphorici dil, ʒijss.
Tinct. belladonnæ, ʒss.
Tinct. digitalis, ʒss.
Syr. aurantii, ʒi.
Aq. chloroformi ad, ʒvj.

One measured tablespoonful in a wineglass of water at 10.30 a.m. and 3 p.m. If considered advisable ♏xii. of tinct. fer. per. may be added to each dose *at the time of taking.* If you find the tonic does not meet the case you might try a little arsenic.

"(3) With care he may go on for some time, but always a handicapped life."

In these, as in all cases of heart defect, the digestive functions have to be carefully looked to.

April 12th, 1915.

Pelvic Abscess following Fowler Position in Appendicitis. — Delatour says that when pyrexia continues, and the wound looks healthy, and careful examination of it shows no undrained pockets, be sure to make a rectal examination, especially if the patient is losing ground, and has frequent bowel movements, with tenesmus and the discharge of mucus. If an abscess is found, do not hesitate to drain it through the rectum. There is apparently little or no danger of infection from this source. To wait for the abscess to be reached easily from in front is to lose valuable time, and the incision through the abdominal wall will leave a weak point for the development of hernia, as drainage must be continued for some time.—*Journ. A.M.A.*, vol. lxiv, p. 857.

THE NEWER INDICATIONS FOR REMOVAL OF THE SPLEEN.

By SIDNEY BOYD, M.S.Lond., F.R.C.S.Eng.,
Surgeon, (Out-patients) Hampstead and North-West London Hospital, Senior Surgeon, Belgrave Hospital for Children, etc.

ALTHOUGH removal of the spleen has been recognised as a surgical procedure since the latter half of the last century, the indications for this operation have, in recent years, been considerably augmented by the discovery that the spleen plays a prominent part in the pathology of many diseases, in which its importance was formerly believed to be only subsidiary. This is all the more gratifying in that many of the diseases in which splenectomy is now indicated are practically uninfluenced by medical treatment, and the results of the operation bid fair to become, in many cases, as brilliant as any in the domain of surgery.

An American writer (1) has recently said: "It is necessary in these, as it is in other medico-surgical conditions, for the physician to learn the details of surgical progress." It is equally necessary for the surgeon to become familiar with the progress made in medicine and pathology, although here, as has often been the case elsewhere, surgical enterprise has gone ahead of pathological knowledge, and, while admitting that the physician should always be consulted in any of the medico-surgical conditions in which splenectomy is under consideration, it is to be devoutly hoped that physicians will be equally ready to call in surgical aid in all cases where there is reason to believe that removal of the spleen will be beneficial to the patient.

I do not propose, in the short time at my disposal, to make any reference to traumatic conditions of the spleen, which call for operation, nor to those pathological conditions of the organs which for many years have been regarded as best treated by splenectomy. Such conditions are wandering spleen, especially when complicated by torsion of the pedicle, tumours, simple and malignant, cysts, and infections, such as tuberculosis, malaria, and syphilis. With regard to the latter group, however, attention may be drawn to a remarkable series of cases, recently published (2), in which splenectomy has been performed for a condition known as Egyptian splenomegaly. This disease is apparently fairly frequently met with in Egypt and its pathology, as far as it is at present known, appears to be somewhat different from splenic anæmia and Banti's disease. Egyptian splenomegaly has a seasonal variation, and is characterised by attacks of fever with enlargement of the spleen, which is progressive and followed at an early stage of the disease by enlargement of the liver, due to multi-lobular cirrhosis, and later on, by ascites. Hæmatemesis is rare and anæmia and leucopenia are usually present.

Splenectomy has been performed with marked success in a considerable number of these cases and has proved that here also, we have an example of a primary disease of the spleen, which is usually fatal if left to pursue its course and in which removal of the organ in an early stage of the illness is a life-saving measure.

It will, of course, not be possible in this short paper to give anything like a full account of the various diseases discussed, but merely to pass in review their more important features and to record the amount of success splenectomy has so far attained in each case.

(1) SPLENIC ANÆMIA.

One of the most important diseases coming within the scope of this paper is what is usually designated "splenic anæmia."

The term "splenic anæmia" has been used in an extremely wide sense to include many varieties of splenomegaly with anæmia which cannot be said to belong to any other known disease. Considerable confusion has therefore arisen, but the term "splenic anæmia" should be rigidly restricted, as suggested by Osler, to cases showing an enlarged spleen with oligocythæmia and a low colour-index, no leucocytosis, but usually the opposite condition —leucopenia—a tendency to gastro-intestinal hæmorrhage (which may be severe) and to pigmentation of the skin. Moreover, the disease is usually one of many years' duration, and in its advanced stage is further characterised by cirrhosis of the liver, ascites and sometimes jaundice, making up the clinical picture generally known as Banti's disease. Rolleston (3) recognises two forms of splenic anæmia, one of which develops in the course of from five to seven years into Banti's disease, and the other, with a much more protracted course,

which does not. The disease affects children as well as adults. Many theories have been suggested for the causation of splenic anæmia, into which it is not necessary for me to enter. Banti (4) himself believes the disease is due to some infecting agent, although his cultural experiments have been negative; and even grafting of a portion of the diseased organ into that of a living dog has given no result. Osler (5) regards the spleen as the primary seat of the disease and advises splenectomy in all cases. Rolleston (6) believes it is due to a chronic infective or toxic process in the spleen, and that the gastric hæmorrhage is due to rupture of dilated vasa brevia secondary to some obstruction in the splenic vein, causing the blood in the vasa brevia to pass in the reverse direction to the normal.

More recently A. G. Gibson (7) has described what he believes to be a streptothrix in the spleen in these cases, but his discovery has not up to the present received general acceptance.

Giffin (8) has drawn attention to the association of gall-bladder disease with splenic anæmia, and found evidence of it in five out of twenty-seven cases at the Mayo Clinic. He regards this as a possible source of infection to which the splenic anæmia is secondary.

Splenectomy, however, as is only to be expected, is far more successful in the early stages of this malady than in the terminal stage. The difference in the mortality of the operation is enormous. In the former it is about 10 per cent; in the latter it is about 50 per cent. or even higher.

The treatment of uncomplicated splenic anæmia by removal of the spleen has now been established on a firm basis and should be performed as soon as the diagnosis is made. Many successful cases have been published in the last few years in the British Isles and abroad. Although remissions may occur in the earlier stages of the disease, the course, though protracted is, apart from operation, invariably towards a fatal isssue.

The ultimate result is also far more likely to be satisfactory if an early splenectomy is performed. Nevertheless, in the five cases of splenectomy for splenic anæmia complicated by cirrhosis at the Mayo Clinic (9) four of the patients were in good health many years afterwards; one died three years later with ascites; and one, who had ascites at the time of operation, was alive and well seven years later.

Kidd (10) refers to a case operated upon in the third stage, where the symptoms returned and proved fatal two years after an apparently successful splenectomy. Perhaps a more lasting result would be obtained by combining splenectomy with omentopexy, as suggested by Tansini (11), and successfully practised by him in two cases.

In cases, however, where the diagnosis of splenic anæmia is complicated by other diseases of a chronic infectious nature, of which the most frequent appears to be nephritis, splenectomy is of questionable value. Out of twenty-seven cases of splenectomy at the Mayo Clinic four were of this nature.

(2) Chronic Acholuric (Hæmolytic) Jaundice.

This subject has attracted considerable attention in recent years and from a surgical stand-point it possesses a great fascination, because it is in some of these cases that splenectomy gives its most brilliant results.

According to Leuret (12), icterus with acholuria may occur in the course of infectious fevers, parasitic affections and intoxications, in icterus neonatorum, and in some cases of myeloid leukæmia, but the term "chronic acholuric jaundice" is generally used to cover a clinical group of cases which some authorities regard as entirely distinct from splenic anæmia, while others see in the group merely varieties of this disease. The main points of difference are that in splenic anæmia there is aplasia of the blood-forming organs and no signs in the urine or elsewhere of increased hæmolysis. In acholuric (hæmolytic) jaundice the blood generally shows evidence of an hæmopoietic reaction on the part of the red marrow as evidenced by the presence of normoblasts, myelocytes, polychrome staining and basophile granulation in the red cells (Banti's hæmopoietic form of "splenomegalie hæmolytique"). In some cases as many as 40 per cent. of the red cells may show the latter change. These characteristics may, however, be absent from the blood (anhæmopoietic form) as in a case recorded by Banti, but in which splenectomy nevertheless cured the patient.

For the present, however, perhaps it is better to regard these cases of acholuric (hæmolytic) jaundice as distinct from splenic anæmia and to classify them under the two headings of acquired and non-acquired.

The latter may be subdivided into congenital and familial, although congenital cases may be familial and familial cases may be, but are not necessarily, congenital. Either familial or congenital cases may or may not be hereditary. All cases show an enlargement of the spleen and a certain amount of anæmia, which may be severe, and very often the red cells show an increased fragility in hypotonic saline solutions. This, however, at any rate in the acquired cases, is by no means always the case, and moreover, Gilbert and Lereboullet (13) deny also that it is a true criterion of the non-acquired cases. The acquired cases are usually very much more definitely ill than the non-acquired cases, some of whom may live to a considerable age and carry on an ordinary vocation. Acquired cases, moreover, seem to progress steadily downhill, and are usually fatal in from six to twelve years, terminating in cirrhosis of the liver and ascites (Lintvarev) (14). At the same time it must be remembered that even the non-acquired cases may proceed to hepatic cirrhosis and ascites as in a case reported by Parkes Weber (15).

Several cases of alcholuric (hæmolytic) jaundice, both of the acquired and non-acquired types, have been subjected to splenectomy, in most cases with a happy result, the jaundice disappearing in a very short time. Many of the acquired cases have an enlarged liver which diminishes in size after splenectomy. At the last January meeting of the Clinical Section of the Royal Society of Medicine I showed an example of the acquired type in which splenectomy had effected a complete cure (16).

An interesting point may be noted with regard to the fragility of the red cells. In some cases the fragility has become normal some months after the operation, while in others it has persisted for years although the patients are apparently in good health.

Banti (17) has written at length on the acquired cases and designates the condition as "splenomegalie hæmolytique," while other writers speak of the condition as "hæmolytic splenomegalic jaundice" (Micheli) (18). In some cases the jaundice may be permanently or intermittently absent, a condition which would be covered by Banti's designation, and for which Parkes Weber (19) has suggested the term "splenomegalic anæmia."

Banti regards the condition as due to four factors:

(1) An hæmolytic agent, the nature and situation of which is unknown.

(2) This agent produces chemical substances which stimulate the spleen to hæmolytic hyper-activity. This hyper-activity reveals itself as an enlargement of the organ.

(3) An anæmia which is the direct consequence of the hyper-hæmolysis and is coincident in degree with the increased icterus and uribilinuria which occur in periods of exacerbation of the disease.

(4) Jaundice, which is also due to the same cause. As already stated, this last feature may be absent or considerably delayed. In one of Banti's cases jaundice did not occur until fourteen years and in Umber's (20) case not until eight years after the onset.

It is by no means certain that the icteric tinge in these cases is due to bile pigment, but may quite possibly be due to the direct products of hæmolysis.

Banti believes that these diseases are due to the stimulatory action of some hæmolytic agent on the macrophages of the hæmolytic organs (erythrophages of von Kölliker and Lintvarev), which are the spleen, liver, lymphatic glands, and marrow of bones, and of these the spleen is by far the most sensitive and the most active. By experiments on animals he finds that hæmolytic agents such as hæmolytic serum, toluylene-diamine, and phenyl-hydrazine have a far greater hæmolytic action on normal animals than on those in which the spleen has been removed. These observations confirm those previously made by William Hunter (21).

Banti does not think the hæmolytic agent is *in* the spleen, but that it produces substances capable of exciting in this organ hyper-active hæmolysis. Hence, removal of the spleen gives a clinical cure. Whatever may be the exact *rôle* which the splenopathy plays in this group of cases, there is no gainsaying the fact that they are rapidly restored to health when the spleen is removed. Theoretically, in grave cases, the agent will be sufficiently powerful to revive the hæmolytic process in the secondary hæmolytic organs (liver, lymphatic glands, and marrow). Splenectomy will then be useless or ameliorate only for a time. As far as I know, however, such relapses have not yet been known to occur.

Sir Bertrand Dawson has drawn attention to the liability of these patients to develop gall-stones, and urges this as an additional reason for removal of the spleen in both acquired and non-acquired types of hæmolytic jaundice.

(*To be concluded.*)

April 12th, 1915.

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We regret that owing to the war the number of pages must be diminished for a time.

WHEN, WHERE, AND HOW TO OPEN THE BOWEL IN CASES OF CHRONIC OBSTRUCTION OF THE LARGE INTESTINE.

By A. ERNEST MAYLARD, M.B., B.S.Lond.,
Surgeon to the Victoria Infirmary, Glasgow.

Let me indicate what is to be understood by chronic obstruction of the large intestine; but before doing so it should be stated what the large intestine is intended to embrace. For this purpose the Basle System of nomenclature is adopted. The large intestine commences at the cæcum, extends up the ascending colon, through the hepatic flexure, the transverse colon, the splenic flexure, the descending colon, the iliac colon (comprising that portion spoken of as the sigmoid), the pelvic colon (comprising that part of the bowel which lies in the pelvis, and is often spoken of as the upper 4″ of the rectum), to the rectum.

By chronic obstruction is meant the effects produced by any mechanical agent which more or less completely inhibits the passage of flatus or fæces. These effects are often of such a nature that the patient does not appear acutely ill. The symptoms are chiefly those of abdominal distension, with possibly visible peristalsis. Occasionally there are colicky spasms, and these may evoke vomiting. The vomit is usually of a bilious character. The patient, as a rule, complains more of a sense of discomfort than of actual distress; and it may be roughly said that the lower down in the large bowel the seat of obstruction the less urgent the symptoms.

When to open?—It must be clearly understood that the classes of cases that are being discussed are those where relief to obstruction is necessary; and where, the obstruction being more or less complete, any radical operation, such as that of excision of a growth—if such be the cause—or

plastic measures for short-circuiting should not be entertained until the immediate effects of the obstruction are properly relieved.

By far the majority of cases here referred to are met with among patients who have passed middle life; and, in most instances, the obstruction is dependent upon malignant disease of some part of the large intestine. It must, however, be borne in mind that obstruction may find its cause in fibrous bands or membranes the result of inflammatory adhesions, compensatory attachments, or defective developmental processes, any of which may result in symptoms which do not serve to distinguish one kind of lesion from the other.

Where to open?—This, then, leads to the second of the queries—Where should the bowel be opened? For it is necessary to ascertain the seat and nature of the lesion before we select the particular segment of the large gut that should be tapped. It is sometimes no easy matter—indeed, it is not infrequently impossible—to say where precisely the lesion is situated. It would, however, carry me further than the intended limits of this article to discuss the various symptoms which might be taken to indicate that the disease is situated at any particular part of the large bowel. I must simply state that failing any clear evidence of the seat of obstruction, as can be ascertained by physical examination or by the symptoms present, our first duty is to open the abdomen by a median incision below the umbilicus, and with the fingers, or even the hand, introduced into the peritoneal cavity to find out the nature and locality of the lesion. This information obtained, we are in a position to decide where the bowel should be opened in order to relieve the block proximal to the obstruction. It sometimes happens, owing, perhaps, to the great distension of the gut, that the exact seat of the obstruction cannot be detected. For instance, it is sometimes quite impossible to detect a malignant ring-stricture of the splenic flexure. When we thus fail we must select a region which we feel sure will tap the distended gut. As a clinical fact of some significance I may here indicate a precaution which should be taken when dealing with cases of great distension of the gut. When opening the peritoneal cavity care must be taken that the distended bowel does not suddenly shoot out from the abdomen. In one such instance the bowel, being relieved of the supporting effects of the abdominal parietes, suddenly burst with a loud explosion and projected liquid fæces several feet across the room. When there is such great distension it is wise, both as a precautionary measure and as a help in diagnosis, to tap the projecting distended loop and withdraw a considerable portion of the contents, closing the aperture and carefully cleansing the part before proceeding further.

Let us now assume that we have discovered the seat of obstruction, and that it is located in the rectum or pelvic colon. The course to be pursued will be determined by what we have been able to ascertain regarding the nature and extent of the disease; whether it is such that we consider a future attempt should be made to remove it, or whether the disease is too extensive to render any subsequent operation advisable. Surgeons differ in their opinions and practices regarding the particular part of the bowel to be opened under the two conditions above referred to. In these few remarks, however, I shall venture to give only such practices as have been executed by myself, stating the reasons that have led me to adopt any particular course.

In the case of* *inoperable disease of the rectum or pelvic colon* an artificial anus is made in the left iliac region, that is to say, the iliac colon is opened. In selecting this region for the permanent "anus" the fæces have passed through the greater part of the large intestine, have become more typically solid and fæcal in character, and have become more amenable to the normal physiological processes whereby the habit of regular evacuations may be cultivated. The better management of the outlet may be open to some question when compared, for instance, with an "anus" in the transverse colon; but taking into account the other advantages, the balance seems to be in favour of the left iliac aperture.

In the case of† *operable disease of the rectum or pelvic colon*, my preference lies in the execution of a right lumbar colostomy, and for this special reason, that the necessary soiling of the parietes by the continuous fæcal discharge is well away from the abdominal incisions subsequently needed

* The terms "inoperable" and "operable" indicate that the disease cannot or can be removed by a subsequent operation.

† Idem.

for the radical operation, so that the skin can be efficiently sterilised beforehand, and the wound equally efficiently guarded from infection afterwards.

In the case of *inoperable disease of the iliac and descending colon and in the region of the splenic flexure*, an artificial anus is made in the transverse colon, that is, through the lower epigastric region. This position for the outlet is not only very accessible and convenient for toilet purposes, but by leaving as much of the colon as possible proximal to the obstruction the fæces are more formed and natural than when coming from an opening in the bowel nearer the ileo-cæcal orifice.

But cases now very frequently occur where we may hope, by a subsequent operation of short-circuiting—as, for instance, by planting the ileum into the pelvic colon—to so divert the fæces into the natural rectal channel that a permanent "anus" proximal to the obstruction may not be necessary. Under such circumstances a right lumbar colostomy should be the seat of election, and for the reasons already given that the artificial aperture is well away from the abdominal incisions subsequently required for the anastomotic operation.

In the case of *operable disease of the iliac and descending colon and in the region of the splenic flexure*, colostomy is performed in the right lumbar region, and the reasons for selecting this locality are the same as those just given in the similar case of the rectum and pelvic colon.

In the case of *inoperable disease of the transverse colon*, either an artificial "anus" should be made in the loin, that is to say, as high up as possible in the ascending colon, or a right lumbar colostomy performed. Which method should be adopted will depend upon whether or not a short-circuiting operation is contemplated later. If such a measure is entertained a colostomy should be performed. In the case of *operable disease of the transverse colon* a right lumbar colostomy should be performed.

In the case of *inoperable disease in the region of the hepatic flexure* either an artificial "anus" or a colostomy should be performed in the right loin, that is to say, the ascending colon should be opened as far away from the cæcum as possible. As to which method will be selected the same reasons will guide as those given in the case of inoperable disease of the transverse colon.

In the case of *operable disease in the region of the hepatic flexure* a colostomy should be performed in the right loin, that is to say, the ascending colon should be opened.

In the case of *inoperable disease in the ascending colon* a cæcostomy should be performed. Nothing should tempt one to make an artificial "anus" in this region, for it is worth running some risk in subsequently short-circuiting the ileum into some part of the colon to prevent the perfectly wretched and distressing condition of a patient with a constant discharge of liquid and irritating fæces The young and inexperienced surgeon is fre quently tempted in chronic intestinal obstruction of the large intestine, wherever located, to open the cæcum, feeling quite rightly that by doing so he is safely certain of tapping a part of the bowel that is proximal to the seat of obstruction. The older and more experienced operator, however, looks upon a cæcostomy as the last measure he wishes to resort to to relieve obstruction, and will not undertake to open that part of the bowel unless driven to do so through necessity. The objections to cæcostomy are twofold. First, there is what has already been referred to, the extremely irritating properties of the semi-fluid fæcal discharge. This discharge is more or less constant, and soon excoriates the surrounding skin. The mucous membrane also tends to bulge, and often forms a distinct hernial protrusion. Secondly, there is the great objection that the opening rarely tends to close, even after the removal of all obstruction, and an operation becomes necessary to close it. This operation may truthfully be described as one of the most difficult and troublesome in intestinal surgery; for it has to be undertaken in a region that it is impossible to properly sterilise, and where inflammation has led to a good deal of matting of parts in the region of the cæcum. To be successful the measures attempted must be radical. Elliptical incisions must embrace the skin around the fæcal orifice, then the cæcum must be freely excised, and an anastomosis effected between the ileum and some part of the bowel distal to the excised segment. This must all be done with due regard to any possible infection of the involved area.

In the case of *operable disease of the ascending colon* cæcostomy must be performed.

In the case of either *inoperable* or *operable dis-*

ease of the cæcum ileostomy must be performed. The same reasons apply here as in the case of the treatment to be adopted in disease of the ascending colon. The objections to the formation of an artificial "anus" are equally as forcible; and even though it be not possible to remove the disease, a subsequent effort will be necessary to plant the ileum into the colon in order to relieve the distressing consequences of a continuous escape of extremely irritating material from the small intestine.

How to open.—It may have been noticed that in stating the operations to be performed the term "colotomy" has not been used. The reason for this is, that it now no longer indicates with sufficient exactness what it really means, or rather, perhaps, what the user of it intends that it should mean. Modern advance in gastro-intestinal surgery demands that every term used for an operation should specifically indicate what precisely the operation connotes. The term "Colotomy" is quite a good one if limited in its practical significance to a similar operation performed elsewhere. Thus, Œsophagotomy, Gastrotomy, Cholecystotomy, Urinocystotomy, etc., mean that the viscus has been opened for the removal of something and then closed. So, Colotomy should signify nothing else than that the colon has been opened to remove something and then closed. But the operations which have been solely mentioned in this article are those which indicate a simple tapping of the bowel with *partial* diversion of its contents outside the abdomen; or a complete division of the bowel with a *total* diversion of the fæces through an external opening. In the former case the operation is termed colostomy, cæcostomy, or ileostomy, that is to say, a method is adopted which in all its details of execution resembles an œsophagostomy, a gastrostomy, a cholecystostomy, etc. In the latter case the operation is an artificial "anus," that is to say, the opening performs the part of the true anus.

In performing colostomy and its kindred representative in the cæcum and ileum care should be taken to secure the bowel wall by a few stitches to the margins of the parietal wall before tapping it, so as to prevent soiling of the peritoneal cavity with the escaping contents of the distended gut. A Paul's tube should be introduced and secured with a purse-string suture. In right lumbar colostomy there is not the same need for such precaution, as the peritoneal cavity is not opened.

In making an artificial "anus" several methods are practised. One, however, of the simplest is to withdraw a loop of colon just far enough to allow of a glass rod being pushed through the mesentery. This rod, resting upon the abdominal parietes, secures the gut and sufficiently applies the bowel to the margins of the abdominal incision that no stitches are needed. The bowel is opened and a Paul's tube fixed into the *proximal* end by a suture which encircles the gut. In the course of a week the projecting loop may be excised, thus leaving two orifices, the one above, the artificial "anus," and the other an opening below, that can be utilised for possibly flushing the diseased segment.

Much might be said about the various methods that have been attempted to obtain control of the "anus"; but when we are dealing with obstruction that needs more or less immediate relief, the simpler and more expeditious the measures we employ the more likely are we to do what for the time is best for our patient.

April 19th, 1915.

Arteriotomy for Embolus in Brachial Artery.—Lundmark's patient was a woman of 40 with chronic heart disease, but compensation had never failed. A short time after a felon, an embolus lodged in the brachial artery, where the deep brachial branches, and it was successfully removed ten hours after the first symptoms. In seven cases of arteriotomy for embolus on record, three proved successful, the interval ranging from three to seven hours. Key's patient was a man of 43, and the embolus was at the fork of the femoral artery and was successfully removed after seven hours. Bauer's patient was a man of 39, and the large embolus at the fork of the aorta was successfully extracted after three hours. The only other successful case in his compilation was reported by Mosny, the embolus being removed from the femoral artery after six hours. In Lundmark's case the embolus extracted from the brachial artery was 3 cm. long and about 6 mm. thick. The embolus usually straddles a bifurcation, and the shorter the interval before the arteriotomy the better the prospects.—*Journ. A.M.A.*, lxiv, p. 951.

THE NEWER INDICATIONS FOR REMOVAL OF THE SPLEEN.

By SIDNEY BOYD, M.S.Lond., F.R.C.S.Eng.,
Surgeon, (Out-patients) Hampstead and North-West London Hospital, Senior Surgeon, Belgrave Hospital for Children, etc.

(*Concluded from p.* 128.)

(3) Pernicious Anæmia.

It is probable that some cases of pernicious anæmia are closely allied to the acquired group of cases of acholuric (hæmolytic) jaundice, especially as in some of the cases of pernicious anæmia there is increased fragility of the red cells.

It is well known that increased hæmolysis occurs in pernicious anæmia, and jaundice with acholuria; and histologically the spleens of pernicious anæmia, hæmolytic jaundice, and those of animals in which excessive hæmolysis has been produced by toluylene-diamine are very much alike. Hence it is only natural to suppose that the removal of the spleen may very favourably affect at least some cases of pernicious anæmia. Acting on this supposition, Eppinger (23) (two cases), Klemperer and Hirschfeld (24) (three cases), Mosse (25) (one case), Decastello (26) (one case) in Germany, Antonelli (27) and Bastianelli (one case) in Italy, and Harpole and Fox (28) in America, have removed the spleen with extraordinarily beneficial results, many of the patients who were previously bed-ridden being restored to a life of activity in a few weeks. In some of the cases the spleen was enlarged, but in many it was normal in size.

Hüber (29) has reported a case which improved temporarily after splenectomy, but at the time of the report the patient seemed to be going downhill again. Perhaps it is important to note that in this case the fragility of the red cells was normal, although it is stated that there was evidence of increased hæmolysis, as indicated by excess of urobilin in the stools. An atypical case of pernicious anæmia, operated upon at the Mayo Clinic (30), survived operation, but was not much benefited by it.

In only one of these cases was the operation fatal, and that from broncho-pneumonia, the patient being markedly bronchitic before the operation was undertaken.

These cases are enough to arouse interest in the treatment by means of splenectomy of an otherwise hopeless condition, and perhaps in the near future it may be possible to distinguish those cases which are likely to be benefited by the operation from those which are not. The operation should certainly be recommended in those cases which show evidence of increased hæmolysis.

Klemperer and Hirschfeld believe that splenectomy in cases of pernicious anæmia is beneficial in two ways—first, by removal of a harmful organ; and, secondly, inasmuch as removal of the spleen directly stimulates the red marrow. This is evidenced by the shower of normoblasts and basophile granular cells which almost invariably appear in the blood after splenectomy. This is the view also taken by Hüber.

Mosse points out that the improvement in the blood in successful cases after splenectomy is of a different type to that which occurs in the natural remissions of the disease, the chief points being that in the former there is an approximation of the colour index to normal and megaloblasts are absent. Apparently in Hüber's case, referred to above, the improvement in the blood was more like that which occurs in a natural remission.

(4) Splenic Anæmia of Infants (Anæmia Pseudo-leukæmica Infantum, von Jaksch's Anæmia).

This is an important group of cases which begins in young children from six months to two years of age, and is characterised by severe anæmia, great enlargement of the spleen and often of the liver. The disease is of quite a different type to the splenic anæmia of adults, which may, however, also occur in children. Many of the cases show evidence of congenital syphilis, and often there are signs of rickets, especially about the head. Examination of the blood shows great diminution in the number of red cells, the presence of normoblasts, some myelocytes and heteromorphous leucocytosis.

They do not, however, respond much, if at all, to anti-syphilitic treatment. Thursfield (31) places the mortality at 40 per cent. It is to be hoped, therefore, that surgery will do something to diminish this. Of course, many cases get well on medical treatment, but if a child with this complaint is going downhill splenectomy should be performed. This

has been done in at least two cases with happy results.

Wolff (32) reports a successful case in a child, æt. 18 months, and French and Turner (33) report a highly successful case in a child, æt. 5 years, who had been ill for over four years. In both these cases the rapidity and extent of the improvement after operation was nothing short of marvellous.

(5) Gaucher's Disease.

This is a rare affection, not hereditary, but liable to affect several members of a family, females six times more often than males, usually beginning before the age of twelve, and characterised by progressive enlargement of the spleen and followed later by progressive enlargement of the liver, yellowish-brown pigmentation of the skin of the face, neck, and hands (*i.e.*, the exposed parts), a simple anæmia with leukopenia and in the later stages a tendency to hæmorrhage, especially from the nose and gums, while ecchymoses are liable to form under the skin. Brill's sign, a pale yellow wedge-shaped infiltration of the nasal side of the sclerotic, is generally present.

The onset is insidious, and patients with this disease may last for many years, the spleen becoming of a prodigious size. Jaundice does not occur, and ascites is rare.

Pathologically, the chief point about Gaucher's disease is that the spleen shows, microscopically, an enormous collection of endothelial cells, so that the condition was formerly spoken of as an endothelioma. The condition, however, is not one of tumour formation, and probably represents a peculiar reaction of the tissue to an infecting or toxic agent. A similar endothelial proliferation is found in fatal cases in the liver, lymphatic glands, and marrow of bones. There is no evidence, however, that they are affected early in the disease.

Splenectomy has been successfully performed several times for this condition, but the value of operation in this disease must be subjected to the test of time. Brill and Mandlebaum say: "Inasmuch as more improvement has resulted from splenectomy than from any other measure, it is our duty to recommend this operation, and to insist that it be performed as soon as the disease is suspected."

Although these authors emphatically state that Gaucher's disease is to be regarded as an entity, it must be noted that at least one of the cases diagnosed clinically as typical splenic anæmia at the Mayo Clinic, and operated on as such, was found to show this endothelial proliferation in the spleen supposed to be typical of Gaucher's disease.

Recently, Erdman and Moorhead (35) have collected ten cases of this disease subjected to operation, two of which were fatal. In their own case the liver, which was enlarged, diminished in size after the operation.

(6) Cirrhosis of the Liver.

It is common knowledge that alcoholic cirrhosis (Laennec's cirrhosis) is frequently associated with enlargement of the spleen. Until recent years, the splenic enlargement has always been regarded as secondary to the hepatic cirrhosis, but there are reasons for believing that in some cases the hepatic condition is unfavourably influenced by the state of the spleen and may even be secondary to and dependent upon it.

Eppinger (36) believes that the spleen plays an important part in Laennec's cirrhosis and thinks splenectomy should be given a trial. He finds an increased destruction of red cells takes place in this disease, as evidenced by an increased amount of urobilin in the urine and stools and a certain amount of anæmia, sometimes with a high colour-index. Among English writers, Kidd (37) is also of opinion that splenectomy should be tried in all cases of cirrhosis of the liver with enlargement of the spleen.

In hypertrophic biliary cirrhosis the spleen is practically always enlarged, and in some cases the splenic enlargement precedes the hepatic enlargement (meta-splenomegalic cirrhosis). Moreover, hypertrophic biliary cirrhosis is known to show a tendency to occur in families, and Gilbert and Lereboullet (38) believe that this disease belongs to the same group as the cases of acholuric (hæmolytic) jaundice previously referred to.

Eppinger has recorded two cases of hypertrophic biliary cirrhosis treated by splenectomy. In one case, a man, æt. 42 years, jaundice had existed for ten years, and the liver and spleen were found to be greatly enlarged. After splenectomy the jaundice disappeared in three weeks, the gums ceased to bleed, and bile and urobilin disappeared from the urine. A second similar case also improved very much after splenectomy. Some cases of cirrhosis of the liver in young people, apart from Banti's

disease, certainly seem to depend upon a primary disease of the spleen, and the term "splenomegalic cirrhosis" may be applied to them. Such a case is the following. Last year I operated upon a young woman, æt. 23 years, with a large spleen and ascites, but no anæmia or leucopenia. At the operation, the liver was found to be in an advanced state of cirrhosis. I removed the spleen and thereby cured the ascites, and the patient is now in apparently good health. The most interesting point about this case was that from the spleen Gibson grew a culture of a non-Gram-staining bacillus, and microscopic preparation of the spleen showed the presence of a streptothrix. A full account of this case will be found in the 'Proceedings of the Royal Society of Medicine,' Clinical Section, for June of last year.

It is by no means clear how the removal of the spleen produces its beneficial effect in cases of cirrhosis with ascites. In some cases the spleen is probably the primary focus of an infective process leading to a chronic peritonitis in addition to hepatic cirrhosis, and the beneficial result of splenectomy is readily understood. Or possibly the spleen may be a kind of back-water of the portal circulation and harbours a quantity of toxic material, leading to ascites in the same way. If the effusion into the peritoneum is due purely to perisplenitis, then splenectomy would obviously cure the ascites. But in my own case at first the fluid re-accumulated after the operation and had to be tapped once before it finally disappeared.

It may be argued that ablation of the spleen mechanically relieves the circulation through the liver. In many of these cases the splenic artery and vein are considerably enlarged, and as Hey Groves (39) has pointed out, the blood passing to the liver along the splenic vein constitutes probably more than half of the total blood in the portal vein. This could not be the explanation in my own case for the objection already noted.

Removal of the spleen is not likely to influence favourably the cirrhosis so much as to lead to the disappearance of the ascites in a few weeks. For these reasons, therefore, I feel that, as regards my own case, the first explanation is the correct one.

(7) Lastly, splenomegaly of uncertain nature, which usually means that it is unassociated with anæmia or any other symptoms. Many of these cases are probably splenic anæmia in an early stage, and the blood should be examined regularly to detect any evidence of this. The patient may suffer no inconvenience, but if the spleen becomes movable, it at once becomes dangerous and should be removed. Furthermore, considerable distress may be caused by dragging pain due to the weight of the enlarged organ and also by perisplenitis. Under these conditions also, therefore, the spleen should be removed.

Some of the doubtful cases may be due to endophlebitis and thrombosis of the splenic vein, but as Rolleston (42) has pointed out, it is extremely difficult to distinguish clinically this condition from true splenic anæmia, although Rodman and Willard (43) could not experimentally produce signs of splenic anæmia in dogs by occluding the splenic vein. However, splenectomy is the correct treatment for both conditions, so that pre-operative diagnosis is not of any great consequence.

While not wishing to advocate that every case of enlarged spleen without anæmia or other symptoms should have the organ removed, such cases should be carefully watched, and if the enlargement of the spleen is progressive, or if the general health is deteriorating without any other known cause, the spleen should be removed. Occasionally the splenomegaly disappears.

It must, however, be understood that many cases occur in which splenomegaly forms a prominent part, but in which the other signs and symptoms do not allow one to place the case in any of the foregoing groups. Moreover, mixed types occur, and the whole picture of the disease may change in the course of time. In such cases, until we know more about the diagnosis and pathology of splenic diseases, the question of splenectomy must be decided on the merits of the individual case.

The mortality of splenectomy varies with the condition for which it is performed, and depends largely upon the ease with which the spleen can be delivered out of the abdomen. If there are no adhesions and the pedicle can be easily secured, the operation is simple. On the other hand, if adhesions to the diaphragm and viscera are extensive, the operation becomes more difficult and dangerous. Speaking generally, one would say that adhesions are far less likely to be present in the early stage of the disease than in the later, so that although in 1908 Johnstone (44) placed the mortality for

splenectomy, excluding traumatic cases and cases of leukæmia, at 11·5 per cent., this figure should certainly be halved if operation is undertaken in the early stages of the various diseases for which it is indicated, and we shall be able to advise splenectomy with as much confidence as we now do most of the common abdominal operations.

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April 12th, 1915.

Latent Infection.—Melchior reports a number of experiences which teach that when a phlegmon, a focus of osteomyelitis, or a suppurating glandular process heals, this is by no means equivalent to a complete cure in the bacteriologic sense. The infection has merely subsided into an inactive stage, a partial immunity. The germ of a recurrence or metastasis is already installed. The conception of a "resting infection" throws light on many cases of so-called predisposition or diathesis, and it also is suggestive for estimation of the effects of trauma.—*Journ. A.M.A.*, lxiv, p. 1037.

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We regret that owing to the war the number of pages must be diminished for a time.

ANTISEPTICISM IN RELATION TO OPHTHALMIC SURGERY.

By PERCY DUNN, F.R.C.S.,

Consulting Ophthalmic Surgeon to the West London Hospital; late Lecturer on Ophthalmology at the West London (Post-Graduate) College.

IN ophthalmic surgery, just as in general surgery, the value of the principles of antisepticism and of asepticism rests upon the attention paid to detail. In this regard the essential factor is thoroughness. So far as the eye is concerned this factor is imperative, inasmuch as the ophthalmic surgeon has to deal with an environment which can never be rendered sterile, namely, the conjunctival sac. From this propitious cultivation medium no fewer than thirty-five various micro-organisms have been isolated; the bulk, however, of these are fortunately non-pathogenic; as such, therefore, they do not imperil a non-septic result of operative procedures, and thus the ophthalmic surgeon is able to benefit from the great principles of Listerism. On the other hand, no ophthalmic surgeon would think of performing an intra-ocular operation with the conjunctiva in a septic state, or even within reach of a septic focus. In this regard attention is always directed to the lacrymal sac. Pus infections, arising therefrom, have always to be guarded against, and when present must be dealt with, even to the extent of excising the sac, before any intra-ocular operation can be performed. But thoroughness in the application of antiseptic principles is not only necessary in ophthalmic operations. It equally applies to many ocular diseases, the septic origin of which is not so readily recognised. A septic condition of the conjunctival sac is always a menace to the eye, irrespective of the disease which may happen to be present. An abrasion of the cornea, for example, may be, and commonly is, a simple matter. But let it be exposed to a pneumococcal infection, and the consequences may be fraught

with the gravest danger, not only to the vision, but also to the eye generally. Acute photophobia and blepharophymosis is not an uncommon association, in young children, with a corneal ulcer. The most obvious feature of such children among the poor is their general uncleanliness; their faces are begrimed with dirt; their hands are similarly filthy; their dirty septic fingers are instinctively used frequently to rub their eyes as a palliative to the pain arising from the ulcered corneal surface. By this means the conjunctival sac speedily becomes infected with septic organisms. The toxin these create acts as a powerful irritant to the ulcer, photophobia follows, and this is accompanied by acute blepharospasm. In olden days these cases were described under the head of "strumous ophthalmia"—the term is incorrect because its implied pathology is wrong. Incidentally, it may be observed that the term "struma," descriptive of a morbid entity, has become obsolete in England, having been expunged from the "index of diseases" authoritatively issued by the Royal College of Physicians.

Corneal ulcers, one of the commonest diseases of the eye among the poor, are mostly the result of mal-nutrition, while the photophobia and blepharospasm, by which in children they are accompanied, are the result of neglect and sepsis. The treatment demanded is directed first against the latter condition. On admission into the hospital, the child is placed under chloroform; the skin of the eyelids, and around the orbit, is thoroughly swabbed with a warm 1 in 2000 solution of chinosol; the lids are then separated, and the conjuctival sac is freely douched with the same solution. In the great majority of the cases the ulceration of the cornea is quite out of proportion to the severity of the symptoms. Usually only a small superficial ulcer is found, the other part of the cornea being sound. Atropine and cocaine drops are next instilled. If the blepharospasm be of acute form the outer canthus may be divided by a snip of a pair of scissors. The dressing consists of a hot chinosol fomentation, to be subsequently repeated every four hours. A generous diet is necessary, and iron and cod liver oil, or quinine and arsenic, may be ordered twice daily, or three-grain doses of extract of thyroid, this being generally indicated. Improvement is commonly rapid under this treatment. As soon as the photophobia diminishes the fomentations may be stopped, and a shade placed low upon the forehead to shield the eyes from direct light. As the improvement continues the child, instead of burying its head in the pillows of its bed, will make efforts furtively to open its eyes to see and to play with its toys. When afterwards recovery becomes complete, it is, however, seldom permanent. Relapses are the rule. The return to the home surroundings of its parents, with the lack of good food, exposes the child again to those evil influences, of which the previous attack was the cause. In such cases convalescent-home treatment is the only thing until the return to normal health has become consolidated.

Another aspect of this necessary thoroughness is illustrated in the treatment of traumatic lesions of the eyeball. There is often, as I believe, a want of care in this regard. A workman comes to the casualty department with a foreign body upon his cornea—a fragment of steel, a piece of coal dust, or some other foreign matter. With a spud, casually taken from the instrument case, the foreign body is removed, under cocaine. A drop of castor oil and atropine is instilled, a pad and bandage are applied, and the patient is sent off. The fact, however, that this treatment commonly ensures an uneventful recovery does not exclude the risks arising from the neglect to observe the rule of antiseptic cleanliness. The corneal epithelium has been destroyed by the foreign body, and in the process of the removal of the latter the traumatism has been increased. Here then we have the cornea exposed to all the risks of septic infection, either directly by means of a septic foreign body, or indirectly through septic matter being conveyed to the conjunctival sac by the dirty fingers of the workman. In respect to this common neglect to cleanse the corneal wound, in contrast, we may recall that in general surgery in wounds of the soft parts, no such thing prevails. The antiseptic cleansing of the wounds is the first principle of treatment, and the observance of that principle is equally essential in respect to all wounds of the eyeball, regardless of their nature. When a case of foreign body on the cornea comes for treatment, a speculum should be inserted, the eye being under the influence of cocaine, and the conjunctival sac douched with a warm chinosol solution (1 : 4000). After the removal of the foreign body, the douching

should be repeated. By this means we have secured, at least as far as possible, a non-septic environment for the corneal wound. Again, just as the general surgeon pays attention to the sterilisation of his instruments previously to operating, so care should be taken to sterilise the spud with which the extraction of the foreign body is effected. It is true that these details may have a trivial aspect and be regarded as unnecessary; at the same time it is equally true that not one of them transcends the principles of wound treatment, upon which the success of modern surgery so largely depends.

But a value belongs to these small and what may be thought to be unnecessary details, apart from their influence upon the treatment of the case. They inculcate a routine in the treatment of ocular wounds, a routine which having once been adopted is not likely to be neglected, in cases of serious injuries to the eye—those in which penetration has occurred. Antiseptic thoroughness here is of such paramount importance that any lack of its observance exposes the eye to the risk of destruction. A penetrating wound, say, of the ciliary region, is exposed to two sources of infection: First from pus organisms, and secondly from that organism still to be isolated, from which results infective cyclitis. By this organism a toxin is created, which, carried by the blood stream, excites a destructive plastic irido-cyclitis in the fellow eye. In order to avoid an infective process in these cases thoroughness in antiseptic detail is imperative, and often the safety of the eye depends upon the observance of this. I am certain that I have saved many eyes by following this necessary rule. A bad case was that of a paviour: He was accidentally struck, while at work in the road-way, with the end of a pick wielded by a fellow workman in front of him. Being in use on the roadway, it is easy to imagine the septic matter adhering to the pick; the point of the pick perforated the lower lid, and either burst or penetrated the patient's right eye, causing a large jagged wound just outside the limbus, on the inner side, followed by extrusion of the lens. The globe was collapsed; the ciliary processes were exposed; blood filled the anterior chamber, and was oozing freely from the wound when I saw him shortly after the accident.

There seemed but little prospect of saving the eye. Nevertheless the eye was thoroughly douched for ten minutes with a warm chinosol solution (1 : 2000), iodoform ointment was applied to the eyelashes, the lid wound attended to, and a chinosol fomentation applied. The eye did well from the beginning and a perfect recovery followed; douching was practised twice daily in the course of the treatment. The recovery was still sound at the end of eighteen months.

Despite the extent of the ciliary wound, no suture of the sclera was inserted. Sutures of the sclera are never necessary. They are unnecessary because scleral wounds heal perfectly satisfactorily without them, as I have always seen. When the globe is collapsed from the escape of vitreous through a scleral wound, the edges of the wound fall naturally together; then repair begins, but long before the contour of the globe has been restored, the scleral wound is strong enough to resist the renewed intra-ocular tension. A scleral suture is a meddlesome form of ophthalmic surgery, based upon a surgical principle, which in this instance does not apply to the eye. There is no such thing as a hernial protrusion of vitreous at the site of a scleral wound which has not been sutured comparable to a ventral hernia after an abdominal section. What purpose, then, does a scleral suture serve? An eyeball slowly recovers its contour, after the loss of vitreous, and the physical result in this regard is equivalent to that observed in a child's rubber ball from which all the air has been expressed through a perforation. The outline of the ball is gradually renewed automatically by air again passing inwards through the perforated opening.

Again, a scleral suture is not only unnecessary, but I believe it to be harmful. It closes the wound in the meshes of which infection may still be retained, whereas as long as the wound remains open it can still be dealt with antiseptically. The only conceivable condition in which a scleral suture might be indicated would be that in which there was loss of scleral tissue. Such a contingency, however, could scarcely arise without the injury to the eye being of such a nature as to preclude any chance of recovery.

A point of which some mention may be made is that in respect to the employment of antiseptic solutions as opposed to those which have no germicidal action for douching purposes in the

treatment of eye cases. The chief effect of all douches is the mechanical one; in the flow of the solution is carried away septic and non-septic matter which may be present. In this respect all douches have the same value, irrespective of their nature. The question, however, is an arguable one, whether after all an antiseptic solution is not better than one having no germicidal properties, so far as ophthalmic surgery is concerned. Of course, there is the objection that antiseptics irritate wound tissues and damage the resisting power of the cells to micro-organisms. On the other hand, the fact remains that they are poisonous to germ life, even if they fail to be directly destructive of it, and in the case of the conjunctival sac they must inhibit the growth and control the virulence of micro-organisms by rendering the environment unfavourable as a culture medium. Non-germicidal solutions do not possess this advantage. If the mechanical effect of these fail the micro-organisms remain with their environment intact. The balance, therefore, of safety is in favour of antiseptic solutions, in the use of which the germicidal effect is added to the mechanical one.

A routine for which I claim some value in intra-ocular operations is to douch the wound after each step in the operation. For this purpose a warm 1 in 4000 chinosol solution is used. In an extraction operation, for example, the wound is douched, using an undine for the purpose, after the section, the iridectomy, the capsulotomy, and the expulsion of the lens. By this means the wound is always kept clear of blood, and assurance maintained that septic invasion is being guarded against.

The last point to which reference may be made is that of the choice of the antiseptic. Here, again, in a matter of detail, ophthalmic differs from general surgery. In this regard one germicide may be mentioned only to be condemned. However useful mercury preparations may be generally for antiseptic purposes, they are quite unsuitable for eye cases. It is impossible to sledge-hammer the conjunctival sac with powerful germicidal solutions with a view to obtaining a direct result from the destruction of the micro-organisms. This method is neither necessary nor is it desirable—unnecessary because of the usefulness of the mechanical effect of douching, undesirable because of the harmful effect of mercury solutions upon the conjunctival and corneal tissues. Some years ago I saw a cornea which had become entirely opaque from the prolonged use of a perchloride solution. Mercury solutions also are held to delay the healing of wounds, and other objections to their use are too well known to require further reference.

Of boracic acid it needs only to be said that the feebleness of its germicidal power renders it practically inert as an antiseptic.

There remains then chinosol. Of this I am able to speak confidently after using it exclusively for more than twenty years. Among its advantages are its non-toxic nature, its ready solubility in water, its powerful efficiency as a germicide, its freedom from causing any ill-effects upon the tissues. It came into use on the continent about twenty-five years ago, Kossmann, of Berlin, claiming for it, among other features, an extraordinary power of penetrating tissues. Chinosol is a coal-tar product, of German preparation, chemically known as potassium oxyquinoline sulphonate, or a neutral compound of oxyquinoline. Some years ago I wrote a short paper drawing attention to its advantages in the treatment of eye cases.* Two points, however, in reference to its use must be borne in mind: first, chinosol solutions oxidise steel instruments; secondly, with solutions above 1 : 4000 in strength some smarting is caused to the eyes. The only other record of its employment in ophthalmic surgery that I have been able to discover is that which appears in McKeown's book 'On Unripe Cataract,' published in 1898. His confirmatory testimony in favour of chinosol is endorsed with some enthusiasm, the measure of which is reflected in the following quotation: "This agent (chinosol) seems to have all the good points of the mercurial salts without their disadvantages. I have abandoned the use of the mercurial salts for a year and a half, and I have been using instead chinosol in a strength of 1 : 4000 for application to the eyeball in operations upon the eye, and likewise extensively in ulcers of the cornea, and occasionally I have injected a solution of 1 : 1000 into the anterior chamber in extensive suppuration of the cornea with hypopyon, and I have not found any objection to it save that it smarts a little. It is my opinion that wounds and ulcers heal more quickly under

* "The Fashion in Antiseptics," 'Med. Press and Circular,' May 5th, 1897.

chinosol treatment than by any other, and with unusually slight opacity." Worthy of note, elsewhere in his book, is the insistence he lays upon thoroughness in regard to the use of the germicide in suppuration of the cornea, whether after operative or other wounds. He writes: "The application of the germicidal solution is carried out very thoroughly and quite differently from what is done at the time of the operation. I am not satisfied with mere instillation, but ensure prolonged contact with the solution. I take advantage of the natural conformation of the parts to give the eye a real bath. The head of the patient is turned slightly towards the other side, while the chin is elevated and the vertex is lowered, causing the eye to be situated in the bottom of a cup. This orbital cup is filled with the solution, and the patient is directed to open his lids, and to keep them open as long as possible. This bath is continued for ten minutes."

The principle of antiseptic thoroughness is essential in the treatment of all ocular wounds, and active infection, when present, can only mean some failure in antiseptic technique, a technique of which thoroughness forms the main feature. And yet in the surgery of the eye no pearl of greater price could have been found than that which Listerism has placed in the hands of the ophthalmic surgeon.

April 26th, 1915.

HUSBAND'S STUDENT'S POCKET PRESCRIBER AND GUIDE TO PRESCRIPTION READING. Revised in accordance with the British Pharmacopœia, 1914. By D. M. MACDONALD, M.D. Fifth Edition. Edinburgh: E. & S. Livingstone. Price 1*s.* 6*d.* net.

We could give no stronger recommendation to this book than to indicate that it has reached its fifth edition. It has the great merit of being small and fitting comfortably into the waistcoat pocket. A happy and most useful feature of this edition is the apt summary of the important changes of the new pharmacopœia, which can be seen at a glance, while the prescriptions containing drugs whose strengths have been altered have in turn been brought into line. It contains prescriptions for most, if not all, the maladies met in everyday practice, and will prove a great boon, especially to the young practitioner.

A CASE OF SPLENO-MEDULLARY LEUKÆMIA WITH PRIAPISM.

By ERNEST C. HADLEY, M.D.; B.S.Lond., F.R.C.S.E., etc.,

Medical Superintendent, North Evington Infirmary, Leicester, and Medical Officer of the Workhouse and Children's Receiving Home, Leicester.

A MINER, æt. 27 years, was at work up till May, 1907, when he met with an accident, in which he sustained a severe injury to his head with a scalp wound, from which there was considerable hæmorrhage. He was never able to return to work again. He was first attended by my father, Dr. Clement Hadley, of Shilton, in June, 1907, who diagnosed "enlargement of the spleen." He was subsequently treated at the Queen's Hospital, Birmingham, and at the Coventry and Warwickshire Hospital until the beginning of 1908, when he again came under the care of my father.

I first saw him on April 21st, 1908, at his own home in consultation, and on several subsequent occasions until his death on May 13th, 1909.

History.—Married, two children, both living and healthy; wife never had any miscarriages; patient's health had always previously been very good; no history of syphilis or other venereal disease; he had never been out of England. Always temperate in his habits, regular at his work, bowels always regular. There was no history of phthisis, malignant disease, or similar blood disease in family.

SYMPTOMS.

(1) *Abdominal enlargement.*—This, as is usual in this complaint, was the first symptom noticed by the patient. He stated that it was on account of this that he was unable to return to work, as he could not stoop or bend because "his stomach was getting so large."

(2) *Continued acute priapism.*—Started April 12th, 1908, and continued until May 25th, 1908, without intermission. The pain was most excruciating and referred to the glans penis and suprapubic region. There was, in consequence, great difficulty in micturition. The penis was mottled blue and red in colour. Continuous applications of ice had been tried for several days without relief being obtained.

(3) *Acute pain and tenderness over cervical verte-*

bræ and back of head.—Commenced April 19th, 1908, and continued until May 19th, 1908.

(4) *Wide dilatation of the pupils.*

(5) *Reflexes.*—Knee-jerks both exaggerated; plantar reflex almost absent; no Babinsky; ankle clonus not present.

(6) *Fainting attacks* and frequent troublesome attacks of vomiting; between these attacks appetite well maintained.

(7) *Temperature.*—Normal throughout illness.

Pathology of Blood (Fig. 1, micro-photograph).

The blood presented many interesting features and was simply packed with *myelocytes* (as micro-photograph shows).

The myelocytes were chiefly the large neutrophile myelocytes (Fig. 2 *a*); they were clumped together in masses of seven or eight or more.

There were also many non-granular myelocytes (Fig. 2 *b*, *c*) and eosinophile myelocytes (Fig. 2 *d*), also some abnormal forms as fractured eosinophile myelocytes (Fig. 2 *e*), and quite a large number of small forms of eosinophiles crowded with granules, some with polymorphar nuclei.

The red corpuscles were various—megalocytes,

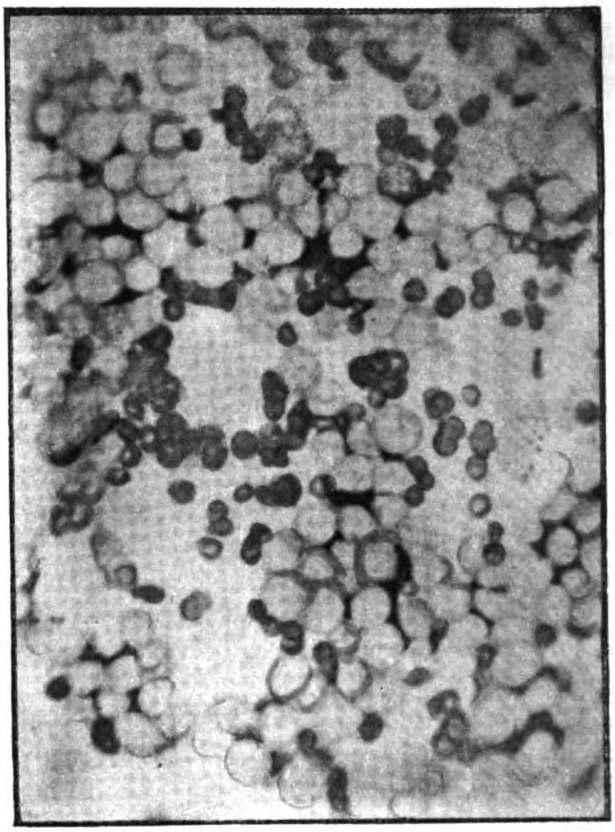

Fig. 1.—Micro-photograph of blood film. × 1000.

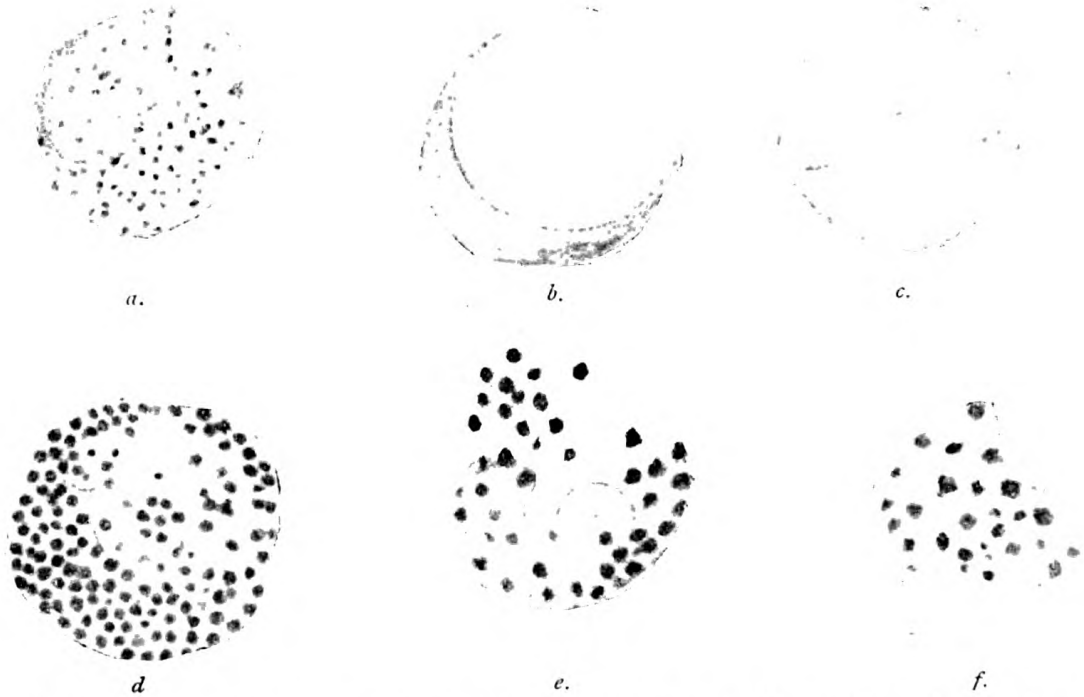

Fig. 2.—Types of the chief varieties of myelocytes found in this case. *a*. Neutrophile myelocyte. *b*, *c*. Non-granular myelocytes. *d*. Coursely granular eosinophile myelocyte. *e*. Fragmentation form of eosinophile myelocyte. *f*. Basophile myelocyte or "mast cell."

poikilocytes, microcytes, and a fair number of normoblasts.

Polymorphonuclear leucocytes were the most abundantly present of the normal leucocytes, but of course relatively quite few; small lymphocytes were very scanty even in proportion to the polymorphonuclear leucocytes.

The proportion of $\frac{W}{R}=\frac{1}{2}$, although in many fields this ratio was reversed.

Three or four "mast cells" (Fig. 2*f*) were frequently seen in a single field.

Subsequent History.

May 13th, 1909: The patient and his relatives insisted upon having an exploratory abdominal operation performed, although it was emphatically pointed out to them that it could do no good, and would probably only hasten death.

The abdomen was opened on the off chance that pressure might be relieved, but only about ½ pint of clear fluid was released from the peritoneal cavity. The sub-peritoneal tissue was full of a clear fluid which raised the peritoneum into blebs.

The spleen was free from adhesions, dark red in colour; it was explored and found to be quite solid.

The patient died shortly after the operation.

The spleen weighed 15 lb.; the liver and other organs were normal to the naked eye.

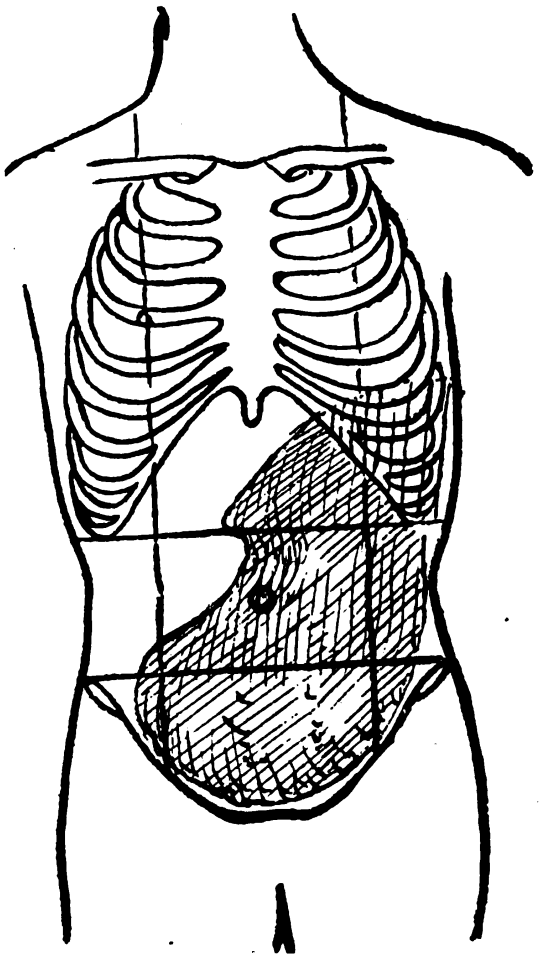

Fig. 3.—Showing shape, position, and size of spleen, when mapped out on the abdominal wall.

Observations upon Causation of Symptoms.

The abdominal enlargement—due to uniform enlargement of the spleen, the spleen coming forward as is usual to occupy a position in front of the intestines, the notch could be distinctly felt and mapped out.

The continuous acute priapism and pain in spine and back of head and altered reflexes. Suggested causation: (i) Clogging of the corpora cavernosa with masses of myelocytes causing an impediment to the proper return of blood and producing a chronic turgescence of the penis.

The sticky nature of the myelocytes is not sufficiently realised.

It is well illustrated in the micro-photograph (Fig 1), in which the myelocytes are seen sticking together in clumps.

The knowledge of this important fact also, I think, explains why ice application failed to relieve the priapism, for would it not tend still further to impede the circulation by constricting the vessels? Warm applications were rather indicated to dilate the vessels and enable the masses of myelocytes to pass along more freely.

(ii) Stoppage of inhibitory impulses to the centre for the erection of the penis by a cause similar to (i) above, that is, clogging up the vessels to that centre with myelocytes. This cause would operate in the same way as that which produces priapism in certain cases of transverse myelitis and injury to the cord.

The exaggeration of knee-jerks and severe pain and tenderness in cervical region and back of head tend to support this theory of causation, though probably both were contributory causes.

Dilatation of pupils explained by the fact that the patient had been taking fairly large doses of hyoscyamus medicinally for some time.

General Observations.

The feature of the case was the symptom of long continued priapism. It is a symptom quite well known, but one which many medical writers fail even to mention. It is a symptom which is pathognomonic of spleno-medullary leukæmia.

It is also interesting to note that an accident preceded the diagnosis of this disease, because trauma, especially when accompanied by much hæmorrhage, is given as a predisposing or determining cause in some medical text-books.

Literature.

"Stricker's case of Priapism." 'Beitrag zur Pathologie und Therapie der Leukæmie Zeitschr. f. In. Medicin,' 1888, Bd. 14.

April 26th, 1915.

A Hand-book of Fevers. By J. Campbell McClure, M.D.Glasgow, Physician to Out-Patients, the French Hospital, and Physician to the Margaret Street Hospital for Consumption and Chest Disease. London: Shaw & Sons, Fetter Lane.

While following in many respects the usual type of text-books on fevers, this book lays special emphasis on treatment. The primary classification into (*a*) fevers of known bacteriology and (*b*) fevers of uncertain bacteriology is followed. Amongst acute diseases attributed to a diet consisting largely of certain cereals beri-beri and pellagra are considered at some length.

In an appendix is detailed the serum treatment of tuberculosis, which contains many valuable hints. The serum used is contra-toxin No. 4 (Mehnarto). In enteric fever, till the fastigium is over, the writer, as a rule, gives only fluid diet; water is allowed freely in quantities of two to three ounces at a time, milk plain, flavoured with coffee or tea, or diluted with barley water. Clear soup is allowed to the extent of a pint in twenty-four hours. The importance of an adequate amount of carbohydrate is insisted on. In regard to hæmorrhage, he regards the amount of blood lost of little value in prognosis. The drug treatment is discussed at length.

Dr. McClure deprecates the fact that whooping-cough is not a notifiable disease, and rightly so. Both it and measles should be on the list, in his opinion, and early hospital treatment given among the poorer classes, especially when segregation is a difficulty or impossible. As might be expected, the author holds no brief for any special drug for whooping-cough. He employs tincture of belladonna, pushed, if necessary, to physiological limits, with or without bromide of soda. To secure sleep he gives half to one drachm of whisky in hot water and sugar towards evening, and continued, if necessary, at intervals of two or three hours. When convulsions are prominent he gives chloral-hydrate by the bowel. Treatment is a strong feature of the book, which is, above all things, practical.

The Extra Pharmacopœia. By Martindale and Westcott. Sixteenth Edition. In two volumes. Vol. 1, 14*s*. net; vol. 2, 7*s*. net. London: H. K. Lewis.

A review of this work irresistibly suggests a contrast. It first appeared in 1883, composed of 312 pages, with 1900 items and approximately 70,000 words. The latest edition is represented by 1630 pages, 12,500 items and one and a quarter million words. From the point of avoirdupois the first edition weighed $5\frac{1}{2}$ oz., the last $1\frac{1}{2}$ lb. When the substance of the book is considered, the contrast is still more marked and serves as the measure of advanced knowledge in matters medical and pharmaceutical. To many there would appear to be strong reasons for combining the two volumes in one. The resulting size would not be a disproportionate book. A vote on the subject of the profession would not be uninteresting. The wisdom of separate books which has marked the last two editions must have commended and justified itself to authors and publishers alike. The volumes follow the lines which have now become established. The first volume deals with the chemical and therapeutic properties of the important drugs, modes of administration, and medicinal uses. The second volume is devoted to the results of analytical and experimental investigation. The highest compliment which can be paid to the book is to state that it has become indispensable to the practitioner who wishes to keep up to date.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

OBSTRUCTION AND ITS VICIOUS CIRCLES.

By JAMIESON B. HURRY, M.A., M.D.

FOR the health of an organism one of the chief requirements is that its conduits be free from obstruction. This applies to its nervous, circulatory, respiratory, digestive, and excretory systems—in brief, to all the innumerable canals which traverse the body. Everywhere does obstruction cause disorder, which varies from temporary discomfort to the destruction of an organ, or even to death.

Obstruction may be due to defective or excessive motive power, to narrowed conduits, or to inspissation of solid or liquid matters in transit, and frequently requires the aid of the *ars medendi.*

The healthy organism possesses numerous mechanisms for the removal of obstruction. Amongst them are coughing, sneezing, vomiting, increased peristalsis, more vigorous cardiac systoles, abdominal straining, and in minor degrees of disorder they prove highly efficient. These mechanisms, however, are of limited potency. They often fail of their purpose, and it is a remarkable fact that such failure usually aggravates the primary disorder and thus establishes a vicious circle.

The following are some common sources of obstruction:

(1) Alteration in motive power: (*a*) Muscular weakness, (*b*) muscular spasm.

(2) Alteration in calibre of channels: (*a*) Changes in the walls of blood-vessels, (*b*) displacement of organs, (*c*) tumours, (*d*) inflammation, (*e*) strangulation, (*f*) effusion.

(3) Inspissation of solids or fluids in transit: (*a*) Inspissation of blood, (*b*) inspissation of fæces.

It will be of interest to point out a few of the circles associated with obstruction, since a familiarity with them is of great assistance in treatment.

(1) Alteration in Motive Power.

(a) *Muscular Weakness.*

Cardiac disease.—Muscular weakness is frequently associated with cardiac failure. Such failure, indeed, causes the gravest possible obstruction.

Two resulting circles may be briefly alluded to. The first is of short circuit, and is made up of the following factors: failure of the myocardium, enfeebled coronary circulation, lessened myocardiac nutrition, and increasing failure. This circle is due to the circumstance that the myocardium is fed by the blood which it circulates and is thus self-dependent.

The second circle has a longer circuit and embraces the great viscera—lungs, liver, intestines, kidneys, and so forth. Cardiac failure causes vascular obstruction in all these organs. The blood returns to the heart laden with impurities, and this condition leads to further weakening of the myocardium.

Constipation.—Constipation dependent on impaired peristalsis supplies a second illustration. The fæcal stasis frequently increases the paresis by which it was primarily caused, and thus renders the condition a self-perpetuating one.

The stasis, moreover, favours the production of bacterial toxins which aggravate both weakness and stasis. Thus Ofenheim writes:

"The question is, what is the effect of the stasis on the bacteria, and, *vice versâ*, of the bacterial toxins on the stasis? That between them exists a vicious circle has repeatedly been proved. Stasis causes bacterial poisons to be formed; these poisons, again, have a paralysing effect on the intestines, and in this way add to the stasis."*

Abductor paralysis.—Fatal obstruction may be due to paralysis of the abductor or posterior crico-arytænoid laryngeal muscles. In health the glottis dilates with every inspiration, and the deeper the inspiration the wider the glottis. Where, however, the abductors are paralysed, inspiration is no longer facilitated by dilation of the glottis, but impeded by increased approximation of the cords, often involving acute dyspnœa. The greater the dyspnœa the greater the approximation of the cords, and *vice versâ*. The sufferer from abductor paralysis has a sword of Damocles suspended over his head.

* 'Proc. Roy. Soc. Med.,' 1913, vol. vi (i), p. 326.

(b) *Muscular Spasm.*

Spasm of the pylorus.—Pyloric spasm associated with gastric stasis and hyperchlorhydria frequently forms a circle.

During normal digestion the pyloric sphincter remains for the most part closed, relaxing at intervals to allow the passage of acid chyme, and then closing again under the influence of the duodenal reflex. When acid chyme has become neutralised in the duodenum the pylorus again relaxes and allows more chyme to pass; the reflex then again becomes active, and so the process continues until the stomach is empty.

In cases of hyperchlorhydria, however, this mechanism is liable to be disturbed. The usual contraction may be replaced by vigorous and persistent spasm of the sphincter, followed by retention of the gastric contents. Such retention then provokes further hyperchlorhydria and so completes the circle. In fact hyperchlorhydria, pyloric spasm, gastric stasis may form an obstinate concatenation of phenomena.

Mathieu and Roux write thus:

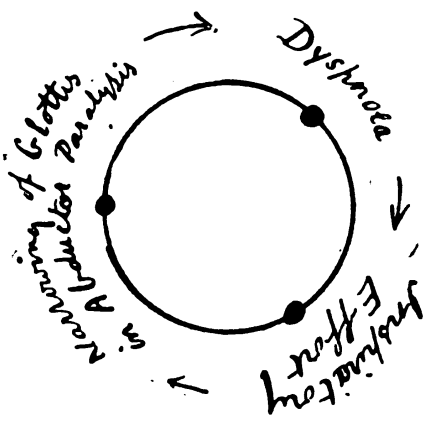
"Reichmann's syndrome presents a remarkable example of a pathogenic vicious circle. The following are the component factors: persistent pain, hyper-secretion of gastric juice, hyperchlorhydria and stasis. The spasm of the pylorus has been proved to be the principal cause of the pain and gastric stasis. The stasis in its turn perpetuates the hyper-secretion, and this again perpetuates the spasm. In a considerable number of cases there is an ulcer at, or near, the pylorus which causes the pain, spasm, and hyper-secretion. In fact the hyper-secretion and stasis prevent the healing of the ulcer." *

Spasm of the colon.—Spasm of the colon is sometimes caused by the irritation of retained fæces, and in its turn promotes further retention and irritation. Especially is such spasm common where the mucous membrane is inflamed.

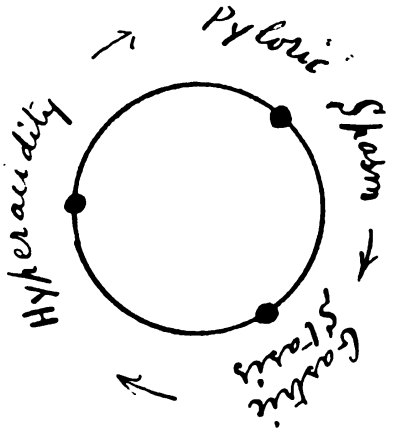
Mathieu and Roux write thus:

"Not only does the spasm cause and keep up the constipation, but the constipation in its turn keeps up the spasm and the colitis."

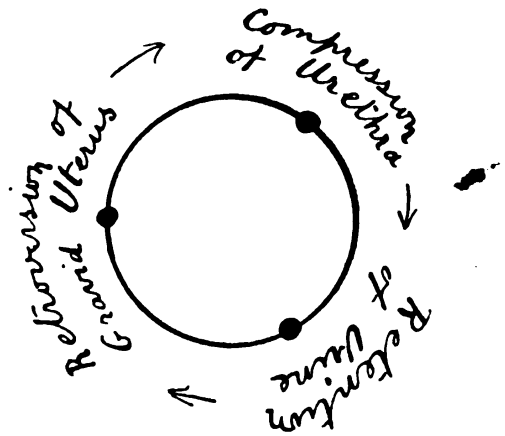
* 'Pathologie Gastro-Intestinale,' series i, 1909, p. 149.



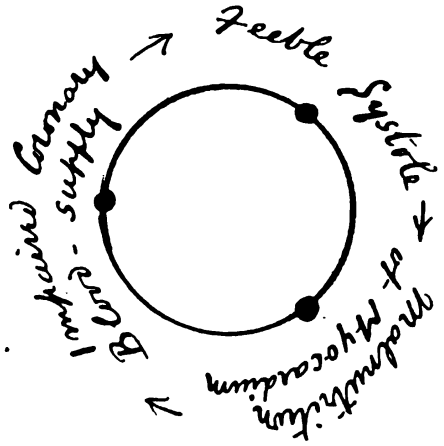
Obstruction due to paralysis.



Obstruction due to spasm.



Obstruction due to displacement.



Obstruction due to sclerosis.

VICIOUS CIRCLES ASSOCIATED WITH OBSTRUCTION.

And again:

"Under the influence of spasm fæcal matters are retained in the intestine, which is thus kept in a state of constant irritation. This irritation then provokes painful manifestations and various reflexes, some of short, some of long circuit. Those of short circuit are confined to the intestinal walls, while the longer ones take a further route and involve the abdominal plexuses. These spasmogenic reflexes in their turn perpetuate the constipation and the intestinal irritation. Thus is formed a vicious circle, which, when once established, shows no tendency to disappear spontaneously." *

* *Ibid.*, series iii, 1911, pp. 93, 303. *Cf.* also series i, 1909, pp. 442, 448.

(2) ALTERATION IN THE CALIBRE OF CHANNELS.

The calibre of the channels of the body is liable to manifold alterations, and these alterations frequently give rise to circles.

(*a*) *Alteration in the Walls of Blood-vessels.*

Arterial sclerosis.—A common condition leading to diminished calibre is sclerosis. Thus the lumen of the coronary arteries may be almost blocked by sclerotic changes, followed by cardiac ischæmia, a weakening of the systole, and further interference with the coronary circulation.

Thus Osler and Macrae write:

"The insufficiency is due to the vicious circle created by lowering the pressure in the coronary

arteries, which occurs directly the ventricle is unable to maintain the required aortic pressure. Thus at the very time when the heart muscle requires the greatest supply of nutrition the supply of blood to the myocardium lessens."*

Varicosity of veins.—Obstruction may also result from the clotting that is often associated with varicose veins. The dilatation leads to increased tension on the vein-walls, and this in turn aggravates the dilatation. The condition is most apt to occur when the valves have become incompetent.

As Noble says:

"It is obvious that as the valves gradually become incompetent, owing to dilatation of the veins, a rise in pressure must follow, and this in its turn leads to further stretching and weakness of the vein-walls, thus establishing a vicious circle."†

Owing to the diminished velocity of the blood-stream, and also possibly owing to the eddies produced by the dilatation, thrombi are apt to form, and to increase as a result of further clotting. The greater the obstruction the larger the surface on which blood can coagulate, and the more rapidly does the thrombus grow.

As Welch says:

"A blood-clot or thrombus in a vessel . . . may itself be looked upon as a foreign body, and lead to further extension of the thrombus. There seems to be a certain self-propagating power in a thrombus."‡

(*b*) *Displacement of Organs.*

The displacement of the organs, such as the colon or the uterus, is another source of obstruction.

Visceroptosis.—The transverse colon may be so displaced as to form an M-shaped loop extending into the pelvis. Such displacement results from and tends to accumulation of fæces, cause and effect reacting on each other. Frequently kinks are also present and aggravate the obstruction.

Thus Pringle writes:

"As soon as any sagging takes place the fixed points, namely, the first stage of the duodenum and the hepatic and splenic flexures, will be dragged on and a certain amount of kinking result. Thus stomach and bowel will have greater difficulty of emptying, there will be retention of their contents, more weight will be thrown on their supports, causing an increase in sagging, and so a vicious circle is set up."*

According to H. M. W. Gray visceroptosis is closely associated with relaxation of the abdominal muscles, thus establishing a further circle:

"The human race suffers many things owing to having adopted the upright posture. The upright posture enters into the formation of another wider vicious circle. Lessened tone affects pretty well all the tissues and functions of the body, but the effect on the flat abdominal muscles is very important. They become relaxed and flabby in action, and their relaxation encourages ptosis of the viscera. The one reacts on the other."†

Retroversion of the gravid uterus.—The gravid uterus may be so retroverted that its cervix compresses the urethra and causes retention of urine. The distended bladder then tilts the uterus still further back and increases the compression.

Pouliot thus described the circle:

"The retroversion, when once complete, causes such retention of urine as in most cases to establish a vicious circle. The distended bladder pushes the uterus further back. The uterus, being tilted into Douglas's pouch, prevents micturition, and the circle can only be broken by an artificial evacuation of the bladder."‡

(*c*) *Tumours.*

Tumours frequently give rise to obstruction which is aggravated by a circle.

Prostatic tumour.—In elderly men the lobes of the prostate may be so enlarged as to encroach on the urethra, when any casual congestion may suffice to cause complete obstruction. The pressure of the accumulated urine in turn aggravates both congestion and obstruction, these two factors reacting on each other.

Desnos and Minet thus describe the process:

"The mechanism of retention is clear. The rapid increase in size of the lateral lobes brings them into contact, the whole gland being compressed by the periprostatic plexuses. Above all the congested and œdematous mucosa forms a plug which obstructs the neck of the bladder. . . .

* 'System of Medicine,' vol. iv, p. 32.

† 'Der Varicöse Symptomen-complex,' p. 54.

‡ Allbutt and Rolleston, 'System of Medicine,' vol. vi, p. 705.

* 'Brit. Med. Journ.,' 1914, vol. i, p. 184.

† Ibid., 1914, vol. i, p. 190.

‡ 'Annales des Maladies Génito-Urinaires,' 1909, p. 5.

Meanwhile urine is accumulating behind the plug and distending the bladder. The pressure of the liquid in this reservoir further aggravates the venous congestion, until the obstruction at the neck of the bladder becomes insuperable."*

In some cases the enlarged lobe of the prostate falls over and closes the urethral orifice like a ball-valve as soon as the bladder contracts. The more vigorous the contraction, the more tightly is the orifice closed; the tighter the closure the greater the contraction.

Cerebral tumour.—Cerebral tumours frequently encroach on the communicating channels of the brain, leading to an accumulation of fluid in the ventricles. The distended ventricles may then so displace the adjacent parts as to increase the obstruction to which the accumulation of fluid was primarily due. For example, the cerebellum and medulla may be pressed down into the foramen magnum so as to partially obstruct that aperture. Such obstruction causes further narrowing of the calibre of the conduits, increases the distention of the ventricles, and renders the condition a self-perpetuating one. Or the dilated ventricles (by compressing the membranes of the nerve roots) may obstruct the lymphatics and veins, such obstruction in its turn leading to a greater accumulation of fluid.

(*d*) *Inflammation.*

Obstruction is frequently caused by inflammation of the mucous membrane lining efferent ducts.

Appendicitis.—Thus the appendix is often the seat of such obstruction, followed by retention and decomposition of secretions. The resulting irritation leads to increased congestion of the mucosa, so that the organ becomes less competent to evacuate its contents. Hence the obstruction is self-perpetuating.

Steward thus writes:

"The lumen of the appendix is so narrow under normal conditions that a very little swelling of the mucous membrane, such as would be produced by a mild degree of catarrh, must inevitably produce some degree of obstruction. This obstruction would result in stagnation from want of drainage, which again would cause a persistence of the irritation and swelling of the mucous membrane. It would appear that this is the way a vicious circle may be set up."*

Hypertrophic rhinitis.—Another illustration occurs in cases of hypertrophic rhinitis.

Yearsley thus describes the condition:

"Defective drainage of the nasal cavities predisposes to microbic infections and their resulting inflammations. Bacteria find a suitable nidus behind a septal deflection, and the result is that recurrent attacks of rhinitis occur, each attack leaving the patient with increased obstruction, and greater tendency to fresh infection; consequently a vicious circle is established of obstruction, defective drainage, rhinitis, increased obstruction. This is the mechanism by which patients with nasal obstructions are so frequently the victims of the common cold. But the results of this vicious circle are more far-reaching than may appear on the surface."†

(*e*) *Strangulation.*

Various tubes of the body are liable to strangulation, the process being complicated by a circle.

Intestinal strangulation.—A common example is represented by a strangulated intestine, the loop of which becomes tense with effused blood and with gas. The tension causes more gut and mesentery to be drawn into the constricting ring, and in their turn to be strangled and distended.

Thus Barnard writes:

"These coils draw their mesentery along with them within the constriction, which in this way becomes tighter and tighter in the vicious circle."‡

The associated meteorism may give rise to a second circle, as Krehl points out:

"It would appear that diminished muscular tonus and insufficient absorption capacity are of much greater importance in the production of tympanites than is an excessive formation of gases. For this reason meteorism is especially marked in peritonitis and acute strangulation. If the intestines once yield to the pressure of gas within them, a vicious circle is established, for this very distention embarrasses their circulation, and so diminishes their ability to absorb gas."§

Tracheal strangulation.—A dangerous obstruction

* 'Maladies des Voies Urinaires,' 1909, p. 411.

* 'The Practitioner,' 1910, vol. i, p. 790.

† *Ibid.*, 1914, vol. ii, p. 503.

‡ 'Contributions to Abdominal Surgery,' p. 5.

§ 'Clinical Pathology,' tr. by Hewlett, p. 324.

may be caused when the goitre compresses the trachea and narrows its lumen. Acute dyspnœa is liable to come on as a result of any exertion that calls the supplementary respiratory muscles into action. For these muscles in contracting press the goitre against the trachea, further diminish the lumen, and thus increase the dyspnœa. The victim is caught in a dangerous vortex, and, unless promptly relieved by operation, dies miserably from self-strangulation.

Fraenkel thus describes the condition:

"The sufferer gets into the clutches of a vicious circle. The greater the want of oxygen, the more vigorous and prolonged the muscular contractions; the more vigorous the latter, the greater the compression of the trachea and the less room for the admission of air." *

(*f*) *Effusion.*

All effusions into the cavities or tubes of the body are liable to cause obstruction, which may then be complicated by a self-perpetuating factor.

Effusion into the pericardium.—Pericardial effusion may serve as an illustration. A case is thus described by Forsyth:

"There can be no doubt that the pericardial effusion was the cause of death, and its fatal effect was due to mechanical interference with the circulation. Six ounces of fluid were rapidly poured out around the heart. The strong fibrous coat of the pericardium was too rigid to relax for its accommodation, and a sac large enough to contain only the heart was now occupied by the heart and effusion. The result was a positive pressure in the pericardium, leading to compression of the thin-walled venæ cavæ and obstructed return of blood to the auricles. The pulmonary veins shared the fate of their neighbours, and their blood was stemmed back until the lung capillaries grew overswollen. This led to œdema of the lungs, and a copious exudation into their alveoli of serum and even blood, the irritation of which stirred up violent attacks of coughing. The coughing raised the intra-thoracic pressure still higher, and the diastolic filling of the heart became a matter of difficulty. This again aggravated the lung condition, and so the vicious circle went round—heart, lung, lung, heart—with the pericardial effusion for its centre."†

* 'Lungenkrankheiten,' p. 8.

† 'Clinical Journal,' vol. xxvii, p. 220.

Effusion into bronchi.—Obstruction to respiration is sometimes due to the effusion of fluid into the respiratory passages. The fluid may consist of pus due to perforation of an empyema. The resulting cough increases the flow of pus, and the pus increases the cough.

Thus Pick and Hecht write:

"The more the patient coughs, the more profusely the pus streams into the bronchi as a result of the expiratory rise in pressure, and such a vicious circle can only end in death." *

(3) Inspissation of Solids or Liquids in Transit.

In order that the various materials in transit through the tubes of the body may progress freely there must not be an undue degree of concentration. Marked divergence from the normal leads to disorder.

(*a*) *Inspissation of Blood.*

A serious form of obstruction is sometimes associated with increased inspissation of the blood and aggravated by the presence of a circle.

The degree of viscosity during health is liable to constant fluctuations dependent on the processes of digestion, respiration, circulation, elimination, etc. Marked deviation from the normal standard is at once put right by a self-regulating mechanism in which oxygenation, rate of blood-stream, secretion of sweat and urine are amongst the most important factors. The result is that the viscosity, like the other physical conditions of the blood, remains fairly constant.

In Clifford Allbutt's words:

"The standard of viscosity, as a vital condition, is so incessantly re-adjusted by the protective harmonies of the organism as to maintain it not only in stability, but also in more or less abiding equilibrium." †

Under some conditions of disease, however, such as heart failure or impaired respiration, the automatic mechanism may be interfered with, and the body is unable to make the usual response to disordered viscosity. Such conditions are usually associated with a great excess of carbonic acid

* 'Clinical Symptomatology,' p. 259.

† "The Viscosity of the Blood," 'Quart. Journ. Med.,' vol. iv, p. 343.

in the blood, and such excess means increased viscosity and increased resistance, these several factors reacting on one another.

Determann thus describes the circle:

"It is clear that, where the self-regulating mechanism is thrown out of order owing to disordered circulation, a kind of vicious circle is present. For the increased viscosity involves increased resistance, and the increased resistance involves increase of carbonic acid, and this again increased viscosity. We have already shown how the body is able to break through the circle.

"On the other hand, we must not forget . . . that when from some cause or other the viscosity diminishes, a circle of favourable conditions is established. For diminished viscosity, at any rate theoretically, accelerates the circulation; this in turn increases the supply of oxygen, and this again further diminishes the viscosity. Moreover, the improved circulation removes some of the water stagnating in the tissues, and in this way again removes one of the impediments to a free circulation." *

(*b*) *Inspissation of Fæces.*

It is well known how readily obstruction of the bowels is brought about by inspissation of the fæces caused by unusual absorption of the liquid constituents. Such obstruction is apt to perpetuate itself.

Thus Mummery writes:

"The longer fæcal material is delayed in its passage along the colon, the harder will it become, owing to the absorption of water by the bowel walls; and the harder it becomes the less easily will it be driven on by peristalsis, so that a vicious circle is soon established." †

THE BREAKING OF THE CIRCLE.

When disease is complicated by the presence of a circle, the first task of the therapeutist is to disentangle from the symptom-complex the dominant factors (maîtres symptômes of Mathieu and Roux) which are acting and reacting on each other, and so perpetuating the disorder. The associated but subsidiary symptoms will subside when such dominant factors have been dealt with.

The next step will be to select the *locus minoris resistentiæ;* if the circle can be broken at one point recovery will commence.

In order to illustrate the procedure we may select one of the circles alluded to above, viz., that in which pyloric spasm, hyperchlorhydria, and gastric stasis form the dominant factors. The clinical picture associated with this circle will probably present numerous symptoms, such as vomiting of sour liquids, emaciation, pain, insomnia, pyrosis, constipation, epigastric tenderness, flatulence, phobias, all of which can be associated with the dominant factors.

The hyperchlorhydria will usually be the factor most readily dealt with. If the excess of hydrochloric acid is neutralised by magnesium oxide or some other alkali, the pyloric spasm which has been maintained by the superacidity will relax. This will allow the contents of the stomach which have been unduly detained, owing to the spasm, to pass the pylorus. As the gastric stasis is thus relieved, the hyperchlorhydria evoked by the stasis will disappear in its turn, and the circle will have been broken. In severe cases antispasmodics and aperients may be used to supplement the action of the antacid.

This example will serve to illustrate the rational therapeutics of a disorder that is complicated by a circle.

Qui bene diagnoscit bene medebitur.

May 3rd, 1915.

* Determann, 'Die Viskosität des Menschlichen Blutes,' p. 80; Clifford Allbutt, 'Quart. Journ. Med.,' vol. iv, p. 355.

† 'Diseases of the Colon,' p. 219.

Meningocele as a Complication of Whooping-cough.—R. Weigert reports the case of an infant, æt. 4 months, who contracted whooping-cough. The attack was not severe, but a meningocele developed at the side of the anterior fontanelle during the paroxysmal stage. There were no convulsions either during whooping-cough or subsequently, and pressure on the tumour produced no effect. The fontanelle gradually became smaller and pushed the tumour back, but at the age of two years and ten months it still admitted the tips of the fingers, and the formation of the tumour could be felt. The delay in the closure of the fontanelle was doubtless due to the meningocele, as no signs of rickets were present.—*Brit. Journ. of Children's Diseases*, xii, p. 24.

Hemiparesis and Hemiplegia as Early Symptoms of Tuberculous Meningitis.—L. W. Sauer reports the following cases:

A girl, æt. 2 years, lost appetite and flesh. Her father developed open phthisis about three months before. After some weeks paresis of the left arm and leg suddenly appeared, which progressed to hemiplegia within three days. The paralysis became less pronounced within the next few days. As there was an epidemic of acute anterior poliomyelitis in the vicinity, this disease was diagnosed. After a few days, however, tonic and clonic convulsions set in, and the sensorium became perturbed. The eyes were rotated to the right, trismus and opisthotonos became marked. The reflexes were exaggerated.

On examination she was well nourished and lay in relatively deep coma. The eyes were half open, the pupils reacted slowly to light; there was slight strabismus, but no facial paralysis. The neck was arched and somewhat stiff. The patellar reflexes were increased. Babinski's sign was negative, there was no ankle clonus, the extremities were spastic, Von Pirquet's test was positive. The cerebro-spinal fluid was clear, lymphocytes predominated, a fibrin network formed on standing, but no tubercle bacilli were found. The child died twenty-two days after the onset of the paresis.

Necropsy.—The cerebral vessels were dilated, and the brain was covered with a thick gelatinous exudate in which were many greyish-yellow specks (miliary tubercles). The ventricles were distended with fluid. Miliary tubercles were scattered throughout both lungs, and there was one conglomerate focus 7 mm. in diameter.

A boy, æt. 3 years, was seen. His father died of phthisis eight months ago. Ten days ago, while apparently well, the child suddenly developed fever, diarrhœa, and vomiting. He was slightly drowsy at the time, but recovered quickly, and was apparently well five days later. Three days ago he fell while playing, and when picked up his mother noticed that he could not lift his right leg, that the right eyelid could not be raised, and that the right angle of the mouth drooped. The voice became nasal during the past day, and this morning the mother noticed that he could not use his right arm.

He was well nourished. Though drowsy, he answered questions promptly. There was slight right facial paralysis (upper and lower). The pupils were equal, and reacted to light and accommodation. The right arm and leg were paretic, the paresis being more pronounced distally. He could walk, but dragged the right leg. Toe-drop was very apparent. The patellar reflex was more lively on the right side. Kernig's sign was negative. There was a right-foot clonus. The spinal fluid was clear, lymphocytes predominated, and there were fifty-four cells to the c.mm.

The hemiparesis developed into hemiplegia, the neck became rigid, Oppenheim and Babinski's signs became positive on the right side. The pupils reacted slowly to light, internal strabismus became marked, and a choked disc was present. The extremities became spastic, and he died comatose.

Necropsy.—On the lower surface of the brain was a fibrino-purulent exudate, more pronounced on the left side. The fluid in the ventricles was cloudy, and contained flakes. There were some tubercles in the pia. There was a hard, lentil-sized focus in the right lung near the bifurcation of the trachea. It was apparently tuberculous, and was surrounded by a zone of hepatised lung-tissue. Near it were two lymphatic glands with hard fibrous capsule and caseous centre. The liver, spleen, kidneys, and peritoneum contained small tubercles.

In both cases the paresis, which rapidly developed into a paralysis, was the first major symptom of meningitis, so that a diagnosis of acute anterior poliomyelitis was for a time plausible, especially in the first case, in which the hemiplegia cleared up for a short time. It is well known that hemiplegia may develop in the course of a tuberculous meningitis, but as far as could be ascertained from the literature, all early hemiplegias were preceded by some other major nervous symptom, such as convulsions, severe headache, or vomiting. In the present cases the first symptom which showed central nervous system involvement was the paralysis. This does not mean that it was the first symptom of the tuberculous meningitis, for in the first case there was a temporary change in disposition, accompanied by loss of appetite and weight.—*Archives of Pedriatics*, p. 902.

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We regret that owing to the war the number of pages must be diminished for a time.

THE CHANGES OF THE POSITION OF THE FOOT DURING LIFE AND THE CALLOSITIES ON THE SOLE ASSOCIATED WITH THEM.

By EDRED M. CORNER, M.C., F.R.C.S.,
Surgeon and Lecturer to St. Thomas's Hospital, and to the Hospital for Sick Children, Great Ormond Street, etc.

ON the soles of our feet is written a good deal of the habits of our life—our podographs. As we spend about one-third of our life on our feet the characters written on our soles become very important. Experience tells us that many are the tales to be read from a study of people's soles; and a foot, like a man, has a life history. Normally the foot undergoes changes according to the age, station, and occupation of the owner. These changes are natural and must not be mistaken for those which are unnatural and abnormal. As a consultant it is constantly brought to my notice how frequently this is done; sometimes by medical men, but far more frequently by mothers, whilst friends and nurses often will not be convinced that the condition of the foot may be regarded as normal under the circumstances.

In asking a patient about the history and the symptoms from which he suffers, he may be untruthful, intentionally or unintentionally; and it may not be possible to find out or even do more than suspect him. But the tale written in the callosities of the sole of his foot can be seen and are not likely to lie. It is imperative for the examiner of a foot to be cognisant with its uses and methods, which will be shown by the callosities on the sole of the foot. The natural changes of position or disposition of the foot during life may be summed up in the following series of pictures. In fact this lecture deals with what may be called "the life history of the foot."

When a child learns to walk it does so with its legs wide apart, and treading on the whole sole of its foot like a flat-footed person. No one point of the sole is more pressed upon than another. In consequence there is no callosity developed, the whole skin of the sole being thick. Should the child be weakly, from whatever cause, the foot becomes more abducted, flattening the arch as it does so. Children vary very much in the rate they grow and learn to walk. A big, strong and heavy child may easily possess a foot which shows all the characters to be seen in that of a weakly; thus the so-called flat feet of children are susceptible to an explanation which shows that the peculiarities of the feet may be a mere exaggeration of the normal due to the special circumstances of the child, *e. g.* heavy child, weakly child, etc. Thus a first chapter in the life history of the foot is easily recognised; a so-called flat foot with a large, broad uncallositied sole exhibiting variations in degree of abduction according to the special circumstances of the child. These variations are due to the child learning to stand and are not seen in children who never stand, *e. g.* some cases of infantile paralysis.

As the child becomes more active it begins to adduct its foot into the active position, and the toes may turn in. And at the same time callosities begin to develop on the inner side of the heel and under the big toe. Commonly this stage can be recognised when the child is 2 to 2½ years old. But just as children vary very much in how quickly they learn to walk (and talk), they vary as much (probably more) in the rate and extent of adducting their feet. It is also noteworthy that the breadth of the tread of the sole has become less, particularly opposite the instep, because as the foot becomes more adducted (and active) the arch of the instep is raised.

The variety of foot just described is often regarded by the doctors and parents as the normal

FIG. 1.—The shaded areas represent the portion in contact with the ground and covered by thickened skin of a foot when a child (up to the age of 2½ years) walks.

for a child, and the first stage of the foot previously described is sent for an opinion on its abnormal and pathological tendencies.

Having become adducted the foot remains so for some years; but as the child grows, increasing in weight, strength, and in length of time it stands, the foot gradually becomes abducted again. This occurs earlier in girls than in boys as they grow heavier—being actually heavier and less strong about 13 years of age—more quickly at first and use higher heels to their foot wear. If only the circumstances of the patient are sufficient (a lot of standing, weakly musculature, etc.), this natural and normal abduction becomes extreme, and pathological flat foot results. This abduction shows itself on the sole of the foot by the development of callosities under the third toe and on the outer side of the heel. In some cases the callosities of adduction (or activity, under the first toe and the inner side of the heel) are present at the time as those of abduction (inactivity, under the third toe and on the outer side of the heel) (Fig. 2). At other times the callosities of abduction are situated more under the fourth and fifth toes, and may extend along the outer margin of the foot.

From this time onward through life the tendency is to lose the adduction callosities and develop abduction callosities, such as that under the third toe.

Callosities also appear on the tips of the toes, particularly under the terminal phalanx of the big toe, where it may appear at 20 years of age. For instance, Fig. 7 represents the callosities on the soles of a golf professional æt. 20. It is noticeable that little remains of adduction callosities, the signs of activity, whilst the abduction callosities, signs of inactivity and of a firm stance, are well marked in spite of his youth.

Under certain circumstances, such as where one foot is amputated, the adduction callosities on the sole of the remaining foot are not lost. The

FIG. 2.—The soles of the feet of a child aged about 3 years, showing (by shading) the thickened skin of the broad tread of previous years (*e.g.* Fig. 1) and the callosities on the inner side of the heel and under the big toe developed as the feet take the "active adducted" position. Callosities of adduction.

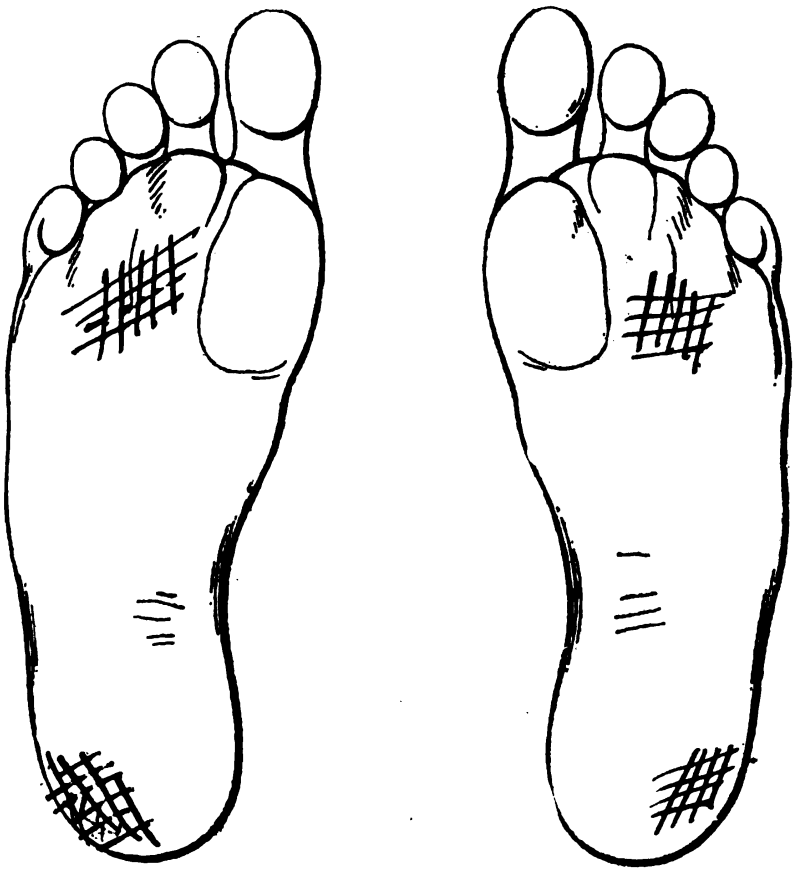

FIG. 3.—Callosities on the sole of the foot of a girl æt. 17 years, illustrating the callosities of abduction.

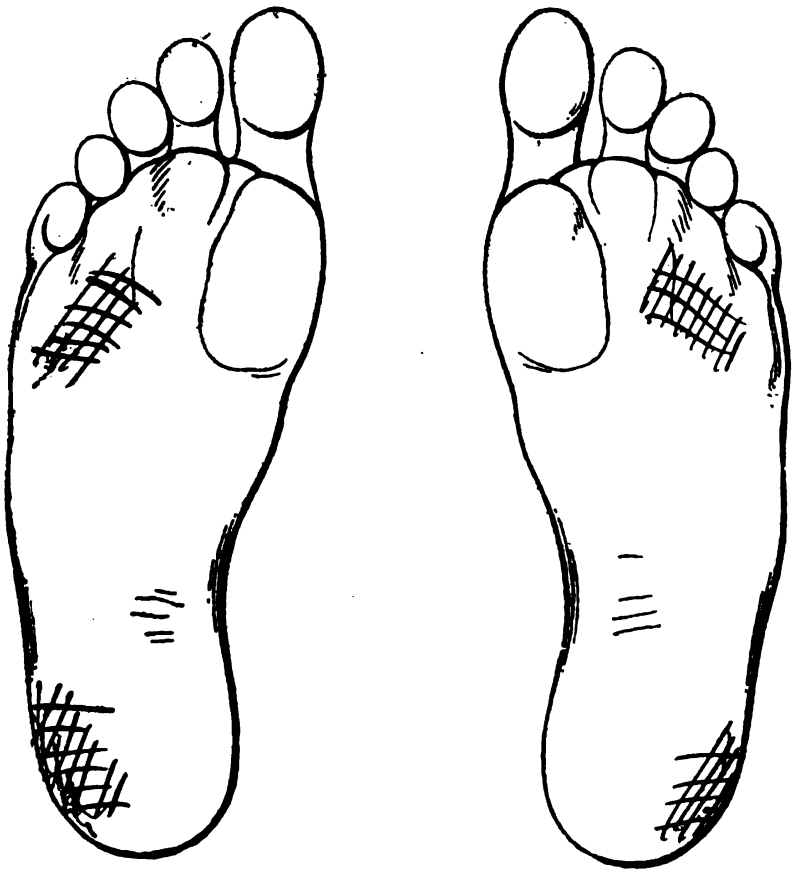

FIG. 4.—Callosities of the sole of the foot of a young man æt. 19 years whose work necessitated much standing. Note that the callosities of abduction are more under the fourth and fifth toes than under the third.

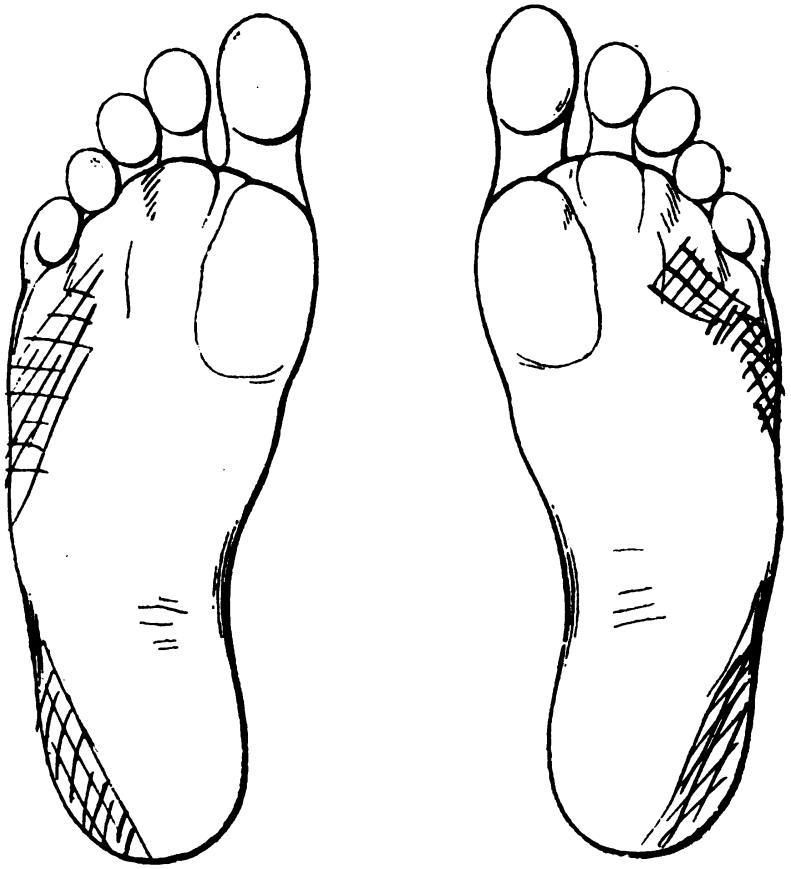

FIG. 5.—The callosities on the sole of the foot are under the fourth and fifth toes, and the callosities under the toes and on the heel tend to spread along the outer margin of the sole.

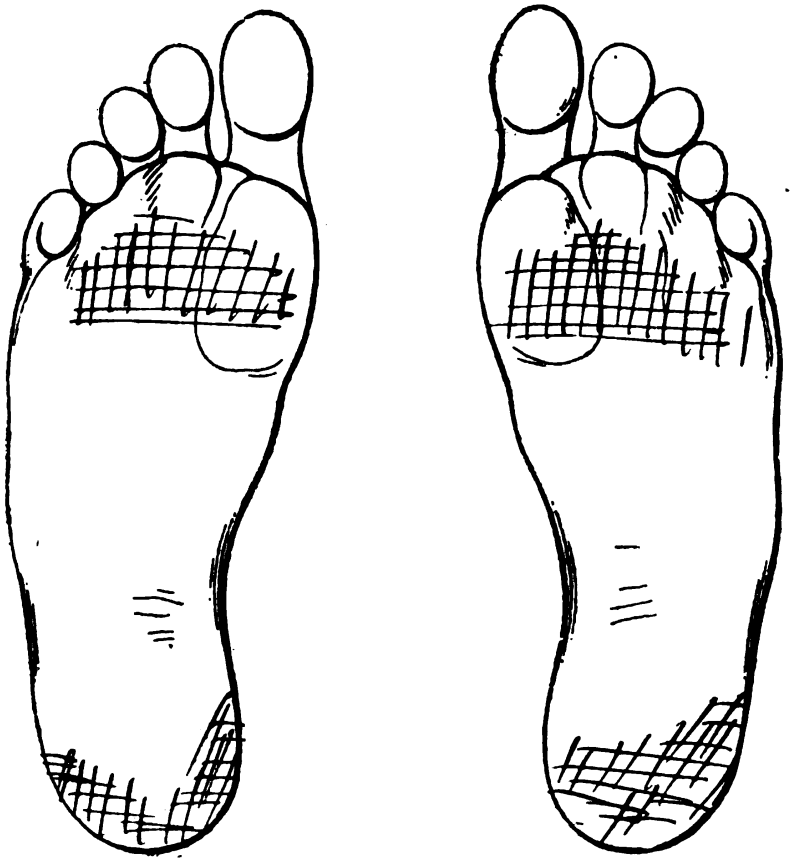

FIG. 6.—Soles of the feet of a man æt. 26 years, showing the callosities of adduction and of abduction.

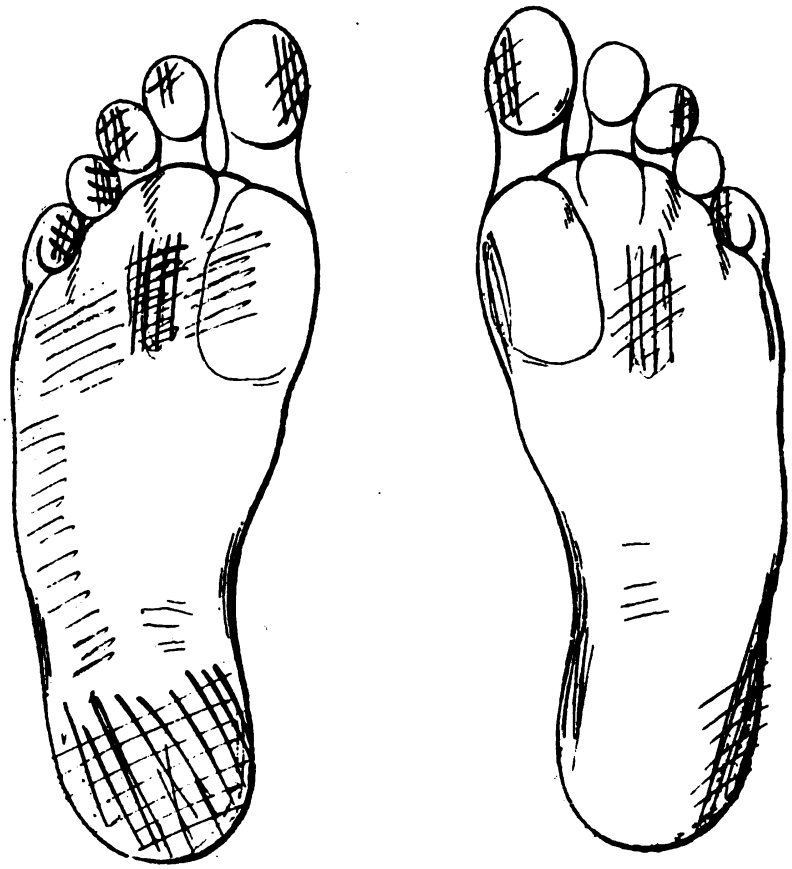

FIG. 7.—Callosities on the soles of a golf professional æt. 19 years, showing the absence of activity callosities and the presence of inactivity callosities, most marked on the sole of the right foot.

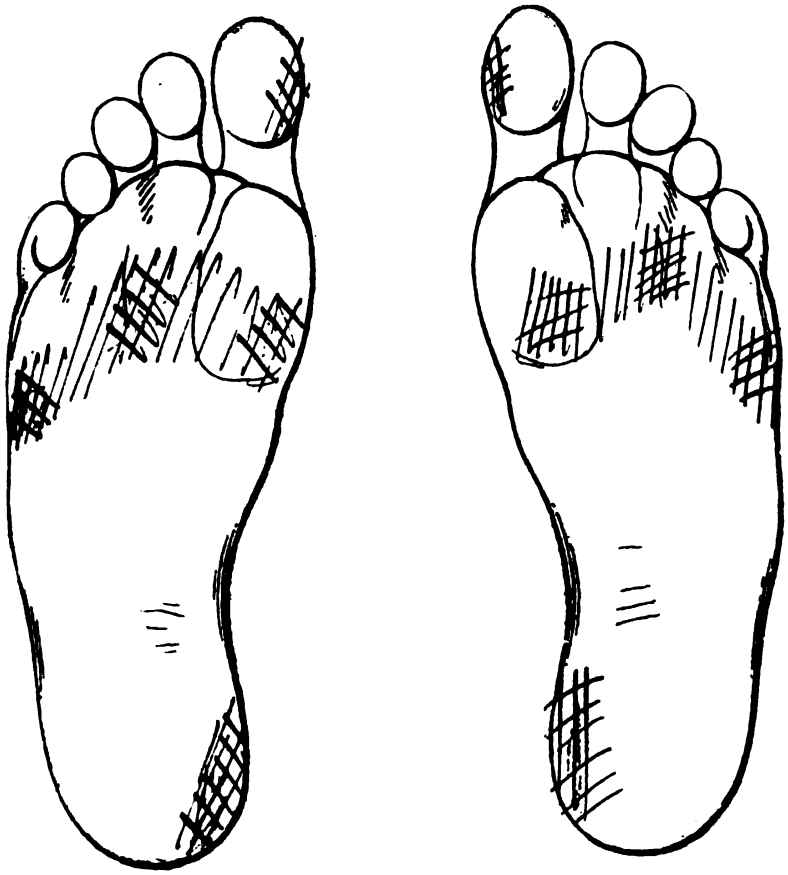

FIG. 8.—Callosities on the sole of the foot of a man æt. 48 years, showing the presence of both adduction and abduction callosities.

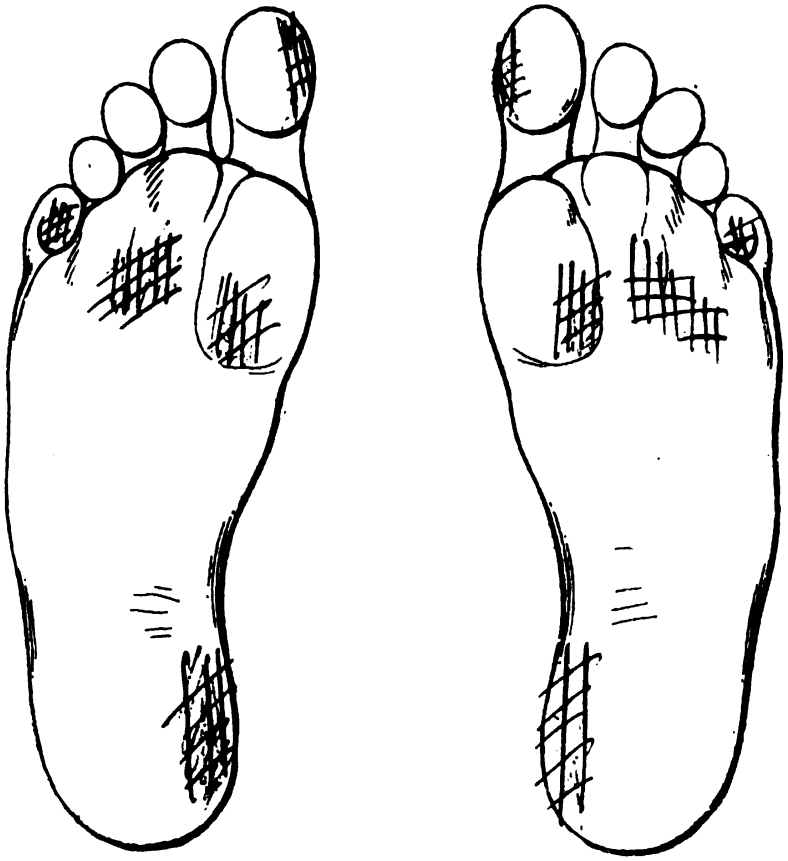

FIG. 9.—Callosities of the sole of the foot of a man æt. 53 years; both adduction and abduction callosities are present.

A B

FIG. 10.——A. The callosities on the right sole of a woman who had lost her left leg. The finer shading represents area of coarse skin. The deeper shading represents callosities. B. The callosities on the left sole of a woman who had lost her right leg. The shading is as in A. In A and B both adduction and abduction callosities are present.

remaining foot has to do double work in activity and bears a lot of the brunt of pressure in inactivity. Hence it shows both adduction and abduction callosities.

It sometimes happens that the feet of an individual are of different sizes. For instance, the left foot of a right-handed person may be broader and longer than the right. The tendency seems to be for the sole of the right foot of a right-handed man to show the callosities of adduction and activity, whilst the left foot shows those of abduction and inactivity. The callosities on the sole of the foot may serve to show when a man is not able to bear the strain of the work he is doing.

Occasionally a callosity is seen under the base of the fifth metatarsal and, if discontinuous with an abduction callosity under the third, fourth and fifth toes, must be regarded as a probable indication of a minor degree of infantile paralysis affecting the peronei muscles.

From the above it can be seen that much information can be derived from the clinical study of the callosities on the sole of the foot. In the progress of a case of flat-foot it will be noticed that many recover by losing the pain in their feet, but a study of the callosities shows that very few get adducted feet. The pain of flat-foot and disability is largely due to an arthritis (osteo-arthritis) of the tarsal joints, secondary to the position of the foot. A few months' rest, as in bed, will cause the callosities of the sole of the foot to disappear.

May 10th, 1915.

A Museum of Fatigue-Eliminating Devices.—In Providence, R. I., a museum of devices for the elimination of unnecessary fatigue has been started. It emphasises the paucity of such devices, for, in spite of the fact that tens of thousands of letters requesting co-operation were sent out and replies indicating willingness to co-operate were received, the museum contains at present only six simple devices, chiefly various types of chairs. One device permits work which has always been done standing to be done sitting. Another device permits the worker to stand part of the time at work which usually had been done sitting. Another type eliminates vibrations from the floor. This is said to have been very successful.—*Journ. A.M.A.*, lxiv, p. 145.

Traumatic Atresia of the Vagina following Labour.—F. Leavitt states that vaginal atresia may be congenital or acquired. The latter is the result of excoriations, abrasions, deeply sloughing ulcers and inflammations of the vagina, accidents and injuries received in childbirth. Very few cases have been recorded. In Allbutt and Playfair's 'System of Gynæcology,' published in 1909, it is stated that "atresia of the vagina following labour is occasionally met with. It is due to sloughing from pressure during labour, followed by cicatricial contraction." It is also stated that "it may be the result of adhesions forming after vaginitis following severe lacerations at childbirth." In the 'Zent. für Gynäkol.' for March, 1909, Sampson reports a "Case of Severe Vaginal Stenosis following Spontaneous Delivery." Leavitt has observed the following case:

An Italian woman, æt. 40 years, had been delivered of three children, who all perished at birth. The last pregnancy was six years ago. The midwife, finding the case unmanageable, retired in favour of a doctor. He in turn asked for help. Ultimately the patient was taken to the hospital, where she was delivered after a long and trying ordeal of a mutilated baby. The woman was a long time in recovering. For several years menstruation had been irregular, the flow growing gradually less, and four months ago ceased. She suffered abdominal as well as pelvic pain at these times.

The vagina was about two inches long, ending in an abrupt *cul-de-sac*. There were evidences of injury to the pelvic floor. Scar tissue could be seen and felt everywhere in and about the vagina. Under anæsthesia the uterus was found large, hard, and fixed. The writer thought of dissecting through the scar tissue and freeing the cervix, but the structures were so dense that he failed even to locate the external os. He did suprapubic hysterectomy. The fundus was free. The adnexa, especially on the right side, were immobilised by adhesions, while the lower segment of the uterus melted into the vault of the vagina. The tube on this side extended high up toward the cæcum, to which the ampulla was firmly joined by the appendix. The fimbriated end of the tube was sealed and distended with retained menstrual fluid. The uterus contained several ounces of black blood. Recovery was uninterrupted.—*Saint Paul Med. Journ.*, p. 124.

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We regret that owing to the war the number of pages must be diminished for a time.

TUBERCULOSIS OF THE RIGHT KNEE-JOINT.*

By LAWRIE McGAVIN, F.R.C.S.Eng.,
Surgeon to the Dreadnought Hospital and to the Hospital for Women; Lecturer on Clinical Surgery in the London (Post Graduate) School of Clinical Medicine.

GENTLEMEN,—The patient whose case I wish you to consider to-day is æt. 28 years, and is suffering from a large and chronic swelling of the right knee-joint. His history is as follows:

Eighteen months ago he developed a swelling over the inner side of the head of the tibia and another at the back of the head of the fibula, and shortly after this a third swelling appeared in the popliteal space. These swellings became tender, the skin over them hyperæmic, fluctuation appeared, and finally they were opened and repeatedly scraped. After some months of treatment they healed up, leaving the scars which you see. During this time the knee-joint was normal, and the patient made no complaint of pain or discomfort. Fourteen months later he began to notice a feeling of weakness in the joint, and gradually a swelling appeared above the patella and on either side of the ligamentum patellæ. Recently this swelling has increased, and he has been unable to walk owing to the pain in the joint, the movements of which have become restricted, flexion being only possible without pain to an angle of about 100°. Pain is complained of, at night especially, of a dull, aching character. There is no history of syphilis or tubercle in his family on either side, and so far as he can remember he has never had any injury to this joint, nor has he ever suffered from any illness. If you examine the joint you will notice that it presents the following characters:

On inspection the knee is very much larger than its fellow, it lies in the fully extended position, the

* Lecture delivered at the Dreadnought Hospital.

skin over it is normal in colour, and the outlines of the swelling do not correspond to the normal outlines of the capsule of the joint. Thus, although the swelling does not rise above the normal limits of the subquadriceps bursa, nor transgress the usual limits of the tibial attachments of the capsule below, it is obvious that at each side, especially at the outer aspect of the joint, there is a very prominent, elongated swelling, reaching upwards over either condyle of the femur. On flexion all of these swellings become more prominent. There is marked wasting of the limb, especially above the femoral condyles, and the calf muscles are wasted and flabby.

On palpation you will find the skin over the joint distinctly warm as compared with that of the other side. The skin is free from the underlying tissues; fluctuation is marked, more especially over the swelling on the outer aspect of the joint. The sensation conveyed to the hand is that of a cavity filled with pulpy material, and a patella tap is obtained with difficulty, although the joint capsule does not feel tensely distended. The patella is freely movable. Pressure gives rise to tenderness only over the inner side of the joint; there is no tenderness over the head of the tibia, the condyles of the femur, nor when interosseous pressure is made in the length of the limb; nor is any grating felt or heard on flexion and extension.

The radiogram shows no alteration in the bones. No other joints are affected. The temperature and pulse are normal, and except for his joint condition the patient has every appearance of perfect health.

I do not propose to waste your time in discussing the differential diagnosis in the case, since, quite apart from his age, history, and the fact of a positive von Pirquet's reaction, I know of no condition which would produce so typically as in this case you see them, the characters of joint tuberculosis. I would rather consider with you the question of the disease as it affects this joint generally, its treatment with special reference to the case before you, and certain points in regard to the now somewhat rare operation of excision of the joint. First of all let us consider roughly the anatomy of the joint.

Of all the large joints in the body this is the most complicated, the most important, the most liable to injury and disease, and the most seriously to be considered from the operative point of view. The following reasons help to explain these facts. It has the largest area of synovial membrane; it is bounded by large masses of cancellous bone on three aspects; it is beset by a perfect retinue of bursæ of which at least two form part of the joint-cavity; it is a box containing accessory cartilages, and its complicated synovial membrane encloses in its meshes a quantity of delicate fatty material; owing to its position it is exposed to a great and constant strain; it is not a ball and socket joint like the hip-joint; and lastly, its great range of movement and the fact of its possessing the power of slight rotatory movement render it particularly liable to strains and intra-articular injuries.

The anatomical points bearing on the question before us which I would have you remember are as follows:

(1) The attachments of the semilunar cartilages · (*a*) both of them by the coronary ligament to the margin of the head of the tibia; (*b*) to each other in front by the transverse ligament; (*c*) the outer to the spine of the tibia; (*d*) inner to the internal lateral ligament, the posterior crucial ligament, and the posterior ligament of Winslow.

(2) The crucial ligaments firmly attached to the spine of the tibia.

(3) The ligamenta alaria and ligamentum mucosum, both running deeply backwards into the joint and somewhat movable within it.

(4) The importance especially of the following bursæ:

(*a*) That beneath the semi-membranosus tendon.

(*b*) That beneath the inner head of the gastrocnemius muscle.

(*c*) That beneath the popliteus tendon.

(5) The intimate relation of the popliteal artery to the posterior surface of ligament of Winslow.

(6) The greater prominence of the internal condyle of the femur and the greater height to which the cartilage rises on the outer condyle.

(7) The attachment of the origins of the gastrocnemius muscle behind each condyle of the femur.

Next I would have you consider the contributory causes of tuberculous disease in this joint. Since tubercle more frequently follows a slight than a severe injury to a joint, it is rare to find it as an immediate sequel of displacement of the semilunar cartilages, but by no means uncommon as an ultimate result of constantly recurring nipping of a cartilage displaced for years. In such cases the disease may originate in one or two ways; it may

commence as a localised deposit on and around the cartilage (usually the internal); or the joint cavity, having been repeatedly distended and the resistance of the synovial membrane generally weakened, the disease may appear in a generalised manner over the whole surface. Again, the disease may originate locally in any of the communicating bursæ of the joint as the result of injuries or sprains of the associated tendons; around the crucial ligaments in the inter-condyloid notch; or in the ligamentum mucosum or ligamenta alaria as the result of nipping of these when enlarged or irregularly formed. Lastly, the presence of a loose body resulting in repeated distension of the joint is at times the cause of generalised tuberculosis. More often, however, as in the present instance, no particular cause can be assigned for the trouble.

It is comparatively rare for the joint tuberculosis of adults to originate in bone: it is essentially synovial from the first; whereas in children the reverse is the case, a fact which helps to explain the earlier tenderness and so the earlier diagnosis in them and possibly also the better results obtained in treatment.

With regard to the present case, let us first settle the question of the possibility or probability of saving the joint. In considering this, I will ask you to do so under the following headings.

Firstly, the character and size of the swelling. As regards its *character*, you will note that true fluctuation is absent; that the sensation conveyed by palpation is that of "lumpiness" and semi-solidity, which would point to the granulation variety of tuberculous disease rather than to the form known as "hydrops articuli." If this is so, you must consider the *size* of the mass. A hydrops articuli may be readily diminished by absorption, but a granulation tuberculosis must be overcome chiefly by conversion into fibroplastic material, but to secure this in so great a mass of material as must be here present is more than can be reasonably expected. Moreover, in so great a mass, it is not unlikely that caseation has already occurred and that the cartilage is affected.

Secondly, is the disease still intra-articular, or has it already transgressed the limits of the capsule?

You will notice, gentlemen, that the elongated prominence along the outer condyle of the femur differs distinctly from the rest of the remainder of the swelling. Here fluctuation is marked; the coverings are thinner, and its upper limit is on a higher level than the normal outline of the joint at this point. It cannot be supposed that this swelling represents a distension of any of the bursæ on the outer side, all of which are comparatively small cavities. It therefore probably represents an extra articular collection of altered synovia and tuberculous *débris*. The swelling on the inner side, on the other hand, is probably the distended semi-membranosus bursa, which is often large and at times reaches considerably higher than the rest of the joint cavity.

Thirdly, the absence of night starting pains, muscular spasm, and any marked rise of temperature is favourable. It is presumptive evidence that the cartilaginous surfaces are so far intact.

Fourthly, the absence of flexion, lateral movement, and of joint affection elsewhere, and the integrity, so far as can be ascertained of his lungs is also in his favour. Thus you will see that were it not for the size and character of the swelling, and the probable extension of the disease to the tissues outside the joint, we might reasonably attempt to deal with the joint by palliative measures.

I would impress points upon you, gentlemen, with a view to urging you not to temporise unwisely with such cases. You are dealing with a disease which, although its progress, it is true, is frequently leisurely, is none the less insidious, deceptive, and relentless. You should have clearly in your minds the evidences which will act as a warning to you that the time for further palliative treatment is drawing to a close, and I would give you the following as being of some practical help.

When, in spite of such treatment, you notice a gradual increase in the size of the swelling; a change from fluid to semi-solid without a concomitant shrinkage; a tendency to slightly increased pyrexia; and an increase in tenderness, you may take it as certain that palliative measures have failed. When you meet with a case which has already developed extra-articular swelling alone; or flexion and rotation associated with night starting pains and pyrexia alone, the indications are almost positively against anything but operation. But supposing that to such untoward conditions is added the presence of discharging sinuses, hectic temperature, pain, and sleeplessness, the indications go beyond excision; they call for

amputation. Indeed, it is by no means certain that in some cases of extra-articular disease without sinuses amputation would not be safer than excision. These, however, must be considered on their individual merits. In the case before you, the reasons why I would advise excision of the joint and why I propose to perform it are these: The patient is a young man, he has a living to earn and which he will earn more easily with a stiff and short leg than with no leg at all; he has no marked pyrexia, no evidence of bone disease, no other tuberculous lesion, and his extra-articular involvement is still comparatively limited. His lesion being extra-articular the time has gone by for palliative treatment, and the indications are not those for amputation, which in the event of the excision failing can still be done as a last resource.

One thing I would urge on you, gentlemen: once having made up your mind that your efforts at palliative treatment are not meeting with success, do not procrastinate. Make up your mind one way or the other and get your excision, erasion, or amputation done. By vacillating delay, often enough well intentioned but foolish, you lay your patient open to an extension of the disease. It may be by invasion of muscle sheaths, by tuberculous osteomyelitis of the bones, by secondary infections through sinuses, or even by generalised tuberculosis.

You will notice that I have not touched on the question of treatment of this case by tuberculin, iodoform, emulsion, or by Bier's hyperæmic method; this I have done purposely, as I consider that none of these methods offer sufficient guarantee of success or are sufficiently rapid in their action. I would class them among the methods of unwise temporisation tending to loss of valuable time, and in such a case as the present their use would condemn the patient to the limbo of lost opportunities.

Now let me touch on some points regarding excision of the joint. I know that certain surgeons would not agree with me that the operation should be done in all cases where the disease is extra-articular, and would urge that many cases have been known to recover under rest and curetting of sinuses. That is quite true; similarly many cases of appendicitis have ended in recovery after repeated attacks without operation. That, however, in my opinion does not in any way counterbalance or justify the loss of life that has occurred in others from the following of this advice. Therefore, I urge you not to postpone excision when, in view of what I have said, excision appears to you to be called for. When, therefore, the time has arrived you will explain fully and honestly to your patient exactly what the operation means to him. It is no good making light of his after condition; he must fully realise that for the rest of his life he must walk with a stiff knee-joint and a limb shorter by at least two inches, probably more, than its fellow; that for this reason his gait must be slower, his foot club-booted, and his wage-earning capacity reduced; but in return he would not require crutches, and he will in all probability be saved a worse fate by removal of his disease. Let us suppose that the matter is arranged; the choice of anæsthetic must be settled. To my mind there can be no possible doubt that spinal analgesia for all such cases takes absolute precedence and for these reasons:

You are faced with what is at best often a long and tedious operation; the loss of blood, even where a tourniquet is used, will, on its removal, be considerable; it should not be favoured in any way, as it certainly would be by the use of ether; the operation is one which involves much shock under general anæsthesia; spinal analgesia at once guarantees your patient very considerable immunity from all these troubles, to say nothing of its greater safety and simplicity. Many of you have had the advantage of judging for yourselves and can, I know, bear me out in this.

A word with regard to the tourniquet. Do not use narrow rubber tubing; such a tourniquet requires to be drawn very tight to control the vessels of the thigh in well-developed patients, and over so long a period is apt to cause bruising and blistering of the skin; the best apparatus for the purpose is a broad Esmarch's bandage. Many incisions have been advised for excision of this joint, and the best is that which gives fullest access to the joint and is the least complicated. I have for long advised and used the direct transverse incision below the patella with a vertical incision along the outer margin of the patella running to the highest point of the sub-quadriceps bursa. I have tried the transverse incision dividing the patella, the H-shaped incision, and the anterior flap method. The first is unnecessary and means

re-wiring of the patella if the bone is to be retained; the second takes longer to suture and interferes needlessly with the blood supply of the flaps; the third gives little access to the sub-quadriceps bursa.

I have also attempted Carleton Flint's method of extra-capsular excision of the joint,* but found it difficult, tedious, and unpractical, in that it does not carry the operation clear of the semi-membranosus bursa.

The incision I advocate gives free and rapid access to every part of the joint. Let me advise you when cutting away the synovial membrane to endeavour to get it out so far as you can in two pieces, that which belongs to the sub-quadriceps bursa, the shaft of the femur and the semi-membranosus bursa, and that which belongs to the tibial portion of the capsule, the sub-gastrocnemius bursæ, and the ligamenta alaria and mucosa, and the crucial ligaments. In doing this cut deeply and with sharp curved scissors, rather sacrificing some of the capsule than leaving synovial tissue behind. At only one point need you exercise special care, viz. over the surface of the posterior ligament of Winslow. Remember the comparatively close association of the popliteal artery to this ligament and, if possible, save the ligament, as being not only safer, but with a view to procuring the strongest tie between the bones. This portion of the joint you will easily reach after division of the crucial ligaments if you fully flex the joint over the closed fist of your assistant placed in the popliteal space. Complete the synovial removal by thoroughly scraping the site of its attachment to the cartilaginous margins and sponging the site over with pure carbolic acid or lysol before making the bone section. Now wash out thoroughly every crevice in the joint. In removing the ends of the bones remember the following points, which make for your patient's subsequent comfort.

The angle formed by the bones when united should be about 175° to 178°. If no angle is allowed the jar of walking is unnecessarily obvious, and if too much is provided the limb is needlessly shortened, and backward displacement of the tibia on the femur is favoured.

The natural greater length of the inner condyle should be preserved so as to allow for the anatomical degree of genu valgum.

The tibia should be divided first as allowing more freedom, when the articular surface is removed, for dealing with the femur, and it should be sectioned from before backwards by a broad amputation saw, not from behind forwards with a frame saw. In carrying this out the leg should be maintained perfectly vertical and the incision made absolutely in the horizontal plane. The femur may then be laid upon the upper end of the tibia for convenience and its articular surface removed, also from before backwards. The amount to be removed must depend on the extent of the disease, and it must be so sectioned as to give proportionately the angle and length of inner condyle already specified. Such of the articular surface for the patella as remains above the saw line must now be removed, by a separate saw-cut, from the face of the femur. The question of *retention of the patella* or its removal must now be considered. To my mind its presence adds but little to the strength of the union, and if its margins show any sign of disease it had better be excised. In any case, if retained its articular cartilage must be removed by the saw, a not very easy matter owing to its buried position.

The *approximation of the bones* and their retention in position is a matter of importance. The approximation of the cancellous surfaces must be accurate and close; the rotation of the tibia must be such as to allow the foot to assume the natural angle when the patient is convalescent. The first of these requirements may be secured by careful splinting, but it is much better if this is assisted by the use of wires, plates or screws. At the same time you must not forget that there must always be some risk of tuberculous infection in thus opening up the cancellous tissue more than is absolutely necessary; the risk is slight, but it must be reckoned with. There are three methods of fixing the bones—they may be wired through their antero-lateral aspects; this will prevent rotation, but not separation by extension of the posterior edges of the bones. A long screw may be passed through either condyle of the femur obliquely downwards into the head of the tibia; this will draw the surfaces firmly together, but it necessitates considerable damage to the cancellous tissue. Lastly, a plate may be used on either side and fixed with Lane's short screws, but it must be a malleable plate, since no plane surface can be

* 'Annals of Surgery,' 1906, i, 426.

obtained here, the surfaces of the femur and tibia meeting in a ridge at the line of union.

By one of these methods, then, the bones are secured, and in the screwing or plating operation the question of the variety of splint is not of such vital importance, but when no fixture or only wire has been used the bones must be firmly splinted. In such a case few splints are better or more efficacious than Howse's excision splint, or the simple padded malleable iron strip known as Golding Bird's excision splint. There is much difference of opinion as to the use of the tourniquet, some surgeons dispensing with it entirely, others advising it and removing it before completion of the operation and tying such vessels as may be caught; on the other hand, my practice has always been that of Sir Henry Howse, who was an undoubted master of the art of excision, viz. to leave the tourniquet in position till firm pressure has been applied over an extensive dressing and then to release it after applying the splint. This is, in my experience, the best of all these methods, for in this operation so large a raw surface is exposed, and so badly placed are the vessels for ligature, that in the endeavour to stop the hæmorrhage much time is lost, much blood is lost, and by the time the wound is sutured, the whole cavity is filled with clot. Whereas in Howse's method comparatively little blood is lost and the period of anæsthesia is sensibly reduced. I have been present on two occasions on which the operation was done without a tourniquet; in both the operation lasted far beyond the usual time allowed, and in both the patient left the table suffering from severe shock, and so blanched as to require saline infusion. With regard to this hæmorrhage, more or less unavoidable in so extensive an interference with the blood-vessels of the joint, it is wise to give calcium lactate for a few days before and after operation. When drainage is employed it should be done from the posterior angle of the wound on either side, and by two separate pieces of rubber tubing. Owing to the laxity of the posterior ligament of the joint, the temptation is to pass a single piece of tubing across in front of this ligament and behind the bones. This, however, is unwise, as it is liable to cause compression of the vessels when the limb is placed on the splint; these tubes should be removed at the end of twenty-four hours, the provisional sutures being then tied.

As should be the rule with all operations on the lower extremity, but more especially in excisions of this joint, the limb should never be covered by the bed-clothes.

In conclusion, gentlemen, you must be aware that excision of the knee-joint is becoming less common every year; it can never be entirely eliminated from operative surgery, as there must in the nature of things be certain cases in which palliative treatment has failed, either from unavoidable circumstances, or unfortunately through ignorance or neglect. I would have you remember, however, that given a patient in good circumstances, no case of early tuberculosis of the knee ought ever to be permitted to progress to the stage of operation. Such an occurrence in these latter days argues little in favour of the knowledge or energy of the medical man in charge of the case.

May 17th, 1915.

A New Type of Ankle Fracture.—F. J. Cotton describes a fracture, the characteristic point of which is backward dislocation with the splitting away of a wedge, large or small, from the back of the tibia. This wedge carries the posterior tibio-astragaloid ligaments, foot, and tibial fragment upwards and backwards. Fracture of the malleoli is associated with this luxation; usually the malleolar fracture is not unlike that of Pott's, though sometimes the internal malleolus and the posterial tibial edge of the joint are included in one piece that is split away in a spiral line. As a rule, however, the posterior tibial fragment is separate, though associated with fracture of both malleoli. As a rule both malleoli are broken; hence the confusion with Pott's fracture.

It has long been recognised that certain cases of so-called "Pott's fracture" are complicated with a backward luxation of the foot. What has *not* been recognised is that nearly all such cases are not Pott's fractures. Of fifty-four cases of ankle fracture with posterior luxation seen by the writer in the last seven years, in only one has the lesion now described not been present—only one case of posterior dislocation with Pott's fracture.

Ankle fractures may be classified as follows:

(*a*) Fibular fractures, above the joint level, first

and commonest (fractures of the external malleolus alone are rare).

(*b*) Pott's fractures of the type Pott described.

(*c*) "Inversion Pott's fractures."

(*d*) The type now described.

(*e*) Fractures just above the joint, and separation of the epiphysis.

The writer's attention was first called to the lesion now described by certain old ankle fractures seen for reconstructive operation—cases that presented extreme backward displacement of the foot. He had not previously met such cases and could find no description to fit them. This lesion has been overlooked and left unreduced by skilled surgeons, despite good radiograms. Not only is it not a simple variant of Pott's fracture, but any attempt to treat it as such ends in serious disaster in a large percentage of cases.

A Pott's fracture or an "inversion Pott's" is bad enough, with the two sides of the articular mortise gone; but when the astragalus is driven back of the mortise and deprived not only of lateral support, but of any vertical bearing, the condition is apt to be hopeless unless properly treated. If the foot and leg rest on the heel, of course the tendency is towards spontaneous reduction, but this occurs only in a portion of the cases. In many the displacement resists even a considerable force directed towards its reduction. Such a case unrelieved, must end in a new joint between the tibia and the forward slant or neck of the astragalus, with limited dorsal flexion, and without any precise support at the sides.

Among the cases sent to the writer for reconstruction were three crippled ankles with backward displacement of the foot of this type. They were repaired fairly well by combining bimalleolar osteotomy (with deliberate forcible rupture of most of the ligament) with preparation of a new concave joint surface of the tibia by chisel and curette (approximately at the normal site). The displaced astragalus was shifted forward into the concavity approximately into its former position. Improved, if not normal, function resulted. After this the writer was on the alert for fresh cases, and is still finding them.

In 1908, a student was brought with an ankle smashed and luxated backward and outward. Obviously, this was the case the writer had been looking for. The fracture of the fibula and the tearing away of the internal malleolus were obvious; the foot was also luxated backward (and outwards as well). Reduction under light anæsthesia was easy. This patient got an almost perfect ankle. The rays showed a wedge from the tibia, then already reduced, that had been displaced backward with the luxated foot; both malleoli were fractured. The other fresh cases were almost identical.

The worst of the displacement is often dealt with before the surgeon sees the case. It is unjustifiable to wait to get pictures before reducing such a painful luxation.

A twist is usually given as a cause of the injury. One patient misstepped the edge of a mat in wrestling; one did the same thing in gymnasium "tumbling"; one twisted her foot outward as she stooped from her chair to pick up a rolling spool, a rug slipping under her foot; another, in walking, twisted her foot out on the edge of a rug that slid, etc. Apparently, an outward and backward twist of the foot is usual, and the fracture is often sustained while the weight is borne on the ball of the foot, not on the flat surface. A disproportionate number of these cases have been encountered in private practice, which shows that the lesion is not, as a rule, the result of industrial accidents.

Reduction is simple if the lesion is understood. All that is called for is forward traction, with the muscles relaxed in moderate plantar flexion. Reduce so, and then lock the reduction in maximum dorsal flexion. Hold in plaster. Results are bad if the lesion is not understood, unless spontaneous reduction has occurred. If reduction, spontaneous or operative, has been accomplished, repair is prompt, and the results are excellent—in no way worse than in Pott's fracture. The after-treatment is that of Pott's fracture, save that early mobilisation is essential, and no weight should be borne on the foot for at least seven or eight weeks.—*Journ. A.M.A.*, vol. lxiv, p. 318.

Plastic Tracheitis and Bronchitis.—Henry Laurie and H. C. Colville record the case of a married woman, æt. 45 years, who was brought to hospital at 11 p.m., suffering from urgent dyspnœa, accompanied by marked whistling stridor. There was cyanosis with retraction of the neck and intercostal spaces. A diagnosis of œdema of the glottis, due to chronic nephritis, having been suggested by her medical man, preparations were made for immediate tracheotomy. But she improved slightly; her colour returned and the retraction lessened, although the whistling stridor was still marked. A more thorough examination was therefore permissible. She had been well up to one week before, when she contracted a "bad cough." This had got worse, until three days before, when the breathing became obstructed, and she gradually became worse. She had always been healthy, except for an attack of rheumatism in the previous winter, with which she was laid up for seven weeks.

The temperature was 99° F., and the pulse 100. The heart-sounds were inaudible. The vesicular murmur was very weak, apparently owing to the small amount of air entering the lungs. On auscultation over the trachea the point of maximum intensity of the stridor was localised over the manubrium sterni, and not over the larynx, as might be expected in œdema of the glottis. There was no œdema in any part of the body. Pulv. jalapæ co., gr. 40, was given, and six leeches, followed by a hot pack, were applied to the front of the neck.

The patient remained much the same for four hours, when she had a severe paroxysm of coughing, and expelled a blood-stained cast about 2 in. long and nearly ¾ in. in diameter. It was cylindrical, and appeared to have been formed in the trachea. The dyspnœa immediately improved. This altered the whole aspect of the case, and the question of diphtheria was immediately raised. A swab taken from the throat was later proved to contain a pure culture of Hoffmann's bacillus. The breathing continued to improve, and in the following night a similar cast was expelled, consisting of a complete mould of the trachea and right and left bronchi. Further improvement followed, and she could now speak in a whisper. The breathing was easy, although some stridor was still present. During the next few days she coughed up small fragments of membrane, similar to the larger ones, and rhonchi were audible over a portion of the anterior surface of the right lung, near its root, indicating the portion of the bronchial tree involved. The expectoration for several days was profuse, and at first more than slightly blood-stained. The expectoration of fibrinous material ceased, the hæmoptysis lessened, and the voice gradually returned. She was discharged about ten days after admission, feeling quite well, except for a little huskiness and slight cough.

Dr. William Ewart opens his article on "Plastic Bronchitis," in Allbutt's 'System of Medicine,' with the words, "This curious and rare disease." In all the classical cases of bronchitis recorded, the condition was confined to the smaller bronchi, "from the tubes of the diameter of a goose-quill (rarely of much larger size) down to the finest ramifications." In the present case, the trachea, both main bronchi, and the larger bronchial tubes on the right side only were affected. The smaller bronchi and bronchioles were absolutely free. This was clearly seen after the expulsion of the second cast, when the stridor was confined exclusively to a comparatively small area about the root of the right lung. Elsewhere throughout the right lung the breath-sounds, though markedly diminished, were perfectly clear.

The only case that Dr. Laurie has seen in any way comparable to this was that of a young man recently admitted from a military camp. The necropsy revealed the trachea, bronchi, and even bronchioles lined with a complete membrane, bacteriologically proved to be diphtheritic. Dr. J. D. Rolleston reported an exactly similar case to the Royal Society of Medicine in October, 1913, in a girl, æt. 5 years.

The ætiology of fibrinous bronchitis is still unsettled. Recent observations point to fibrinous exudations being only found in cases with a high calcium content of the blood.

The natural cure of fibrinous exudations seems to consist in their separation and expulsion in profuse secretion from the part affected. Treatment in this case consisted of small doses of iodide of potassium and large doses of citrate of sodium, both of which diminish the coagulability of the blood and increase secretion from mucous surfaces. —*Med. Journ. of Australia*, p. 145.

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We regret that owing to the war the number of pages must be diminished for a time.

PINE-WOOD SAWDUST AS A SURGICAL DRESSING.

By RUSHTON PARKER, M.B., B.S., F.R.C.S.,

Professor of Surgery in the University of Liverpool; Lt.-Col. 1st Western General Hospital R.A.M.C. (T.F.).

FOR many years I have had the impression that the first use of this substance as a surgical dressing was suggested by a country practitioner, but repeated search has failed to confirm the impression, which, therefore, may not be correct. All the published allusions that I can find trace it to Surgeon-Major Joshua Henry Porter, who noticed the absorbing power of sawdust as used in the "dripping trays" used of old in operating rooms, and who was also struck by its beneficial use in the packing and preservation of imported grapes.

In the 'Dublin Journal of Medical Science' for February, 1877, p. 208, Porter brings it forward in the form of pads enclosed in gauze, and composed of pine-wood sawdust, not only for cases discharging offensive pus, but also as convenient supports in cases of fracture, etc.

The same year in the 'Lancet' of June 23rd, Mr. G. W. Callender of St. Bartholomew's Hospital stated that he adopted it on the advice of Sir Joseph Fayrer, and reported cases in point.

In 1883, in the 'Lancet' of September 22nd, p. 494, the use of carbolised sawdust was clearly and practically advocated by Mr. H. P. Symonds, who used it in the Radcliffe Infirmary at Oxford, and in private practice.

In 1884 in the 'New York Medical Journal' of September 6th, there is reported an address by Dr. Lewis S. Pilcher, of Brooklyn, from which I quote as follows:

"In this cheap and despised material we have likewise a very superior substance for making absorbent wound-dressings. Compared with the

other wood preparations that have been introduced —*i. e.*, wood-wool and wood-flour—I think it is to be preferred, for it is practically as absorbent, and is much cheaper and more readily obtained. The fact . . . leads me to think that it is destined to become the most universally used of all dressing materials in general practice."

A remarkable paragraph in this address refers to Professor Esmarch, who did a good deal to simplify the treatment of the wounded in war. Dr. Pilcher received a little packet consisting of Esmarch's first dressing for the wounded on the battle-field (well-known ever since). This contained originally a triangular and a roller bandage, with pins and two tampons of salicylic jute, enclosed in salicylic gauze; but on the occasion in question the jute of the tampons was replaced by sawdust, treated with corrosive sublimate, the whole being protected in an envelope of parchment paper ready for use. Dr. Pilcher adds, "I show it here as an example of the use to which sawdust is being put in the matter of wound dressings." Part of this address is quoted in Braithwaite's 'Retrospect' for 1885, vol. 1.

In the 'British Medical Journal' for 1887, April 16th, p. 836, is a brief notice of its use by Mr. Hugh Owen Thomas, of Liverpool, through whom many surgeons, including myself, further learnt its value and extensively employed it.

Perhaps the best and most comprehensive, as well as recent and accessible account of the preparation and use of the material, in military as well as in civil conditions, is that by Mr. Charles W. Cathcart, of Edinburgh, in the 'British Medical Journal' of October, 17th, 1914, and to this the reader may be referred for valuable details. He also refers to its use by H. O. Thomas, who had it prepared by the sackfull, and used it under every variety of wound and sore on a very large scale. A similar supply was made for me in the Liverpool Royal Infirmary, and kept on hand in the dispensary by Mr. Prosper Marsden. There I used it for thirty years or more up to 1908, and since in private.

At the time of Mr. Cathcart's paper I gladly wrote in confirmation of the opinion and practice so well expressed by him, as did Mr. F. D. Bennett, of London, who had experience of the use of the material in a hospital on the works at the building of the Manchester Ship Canal. Our two notes appeared in the 'British Medical Journal' of October 31st, 1914.

Some quantities have already been obtained and used in English hospitals in France, and if larger supplies should be sent, and be generally used for a first dressing, its efficacy could not fail to be appreciated by the surgeons and the wounded abroad, as it has already been by my colleagues in the 1st Western Hospital, Fazakerley, Liverpool, and by Lieut.-Col. Cathcart himself in the 2nd Scottish at Edinburgh.

In recent wounds the use of prepared sawdust is almost infinite. It may be enclosed in bags of muslin as used by Porter, Cathcart and others, but I have preferred to put it loose in a sheet of rag, and pin the latter round the part so as when possible not to shed the sawdust in the bed. I have always used it thus perchloridised, and in the above paper briefly mentioned three typical cases of its various utility. These I repeat here, and add a fourth which, in chronological order, now comes second.

I. Sent into hospital by Dr. E. P. P. Machloghlin of Wigan, in March, 1887. A case of perineal lithotomy in a boy, æt. 11 years. He was dressed with sublimate gauze and T-bandage for the first six days, after which a large draw-sheet, folded several times into a triangle, was pinned round the hips and between the thighs, as is done with a baby's napkin, enclosing plenty of the loose sawdust against the buttocks and genitals. The neck of the bladder must have been much dilated, as the mucous membrane pouted for a moment in the wound as one sees in a mare's vagina after micturition. For this reason no tube was put in, the urine draining direct into the sawdust, of which only the wet portions required to be changed and no washing of the part needed. He began to pass urine by the urethra on the eighth day, and ceased to pass it also by the wound on the seventeenth.

II. Sent into hospital by Dr. Owen Bowen, of Liverpool, in June, 1887. A man, æt. 53 years, suffering from fever and prostration, having a boggy cellulitis of the perinæum and right groin, resembling in some respects the worst cases of extravasation, but in reality not connected with the urethra or bladder. Through a hole in the skin a black fœtid slough peeped, and shortly after admission free incisions were made under chloroform up to the right inguinal region, the gangrenous

connective tissue being cut, scraped, and washed away with sublimate solution. A large-sized lithotomy staff was easily passed, and along it the bladder emptied itself of clear sweet urine in the course of the operation. Eucalyptus ointment was freely rubbed into the wound, and perchloridised sawdust enclosed in a triangular napkin pinned on as in the previous case. But the morbid process continued to spread up the abdomen, and it was realised that the incisions and washing, though free, were insufficient, as he continued feverish and delirious.

The incisions and cleansing were extended, under chloroform, on the fifth day. On the sixth the anæsthetic was again given, and still without satisfaction to me, so, about half an hour later I had him conveyed, under chloroform, to a hot bath containing about 100 gr. of perchloride of mercury in about six gallons of water, and then freely rubbed the wounds with my hands while under the liquid, and also incised further until I got well beyond the infiltrated areas and reddened edges of the skin. His temperature dropped that day from 102° F. to 99° F. There was abundant discharge of serum, still foul smelling, and the dressing was renewed three times that night by a special nurse put to him, and he was given milk and soda-water and some brandy.

Meanwhile I had against me the matron and other officials, who wanted him removed on account of the noxious odours and the trouble attending his case. This I firmly refused to permit, and was gratified to find the spread of disease arrested, with definite slight improvement in his condition. But when, on the thirteenth day, his wife stated her intention to take him home I had, however reluctantly, to consent, and gave her a keg of prepared sawdust, enjoining her to use it regularly and call for more whenever she could do with it.

When asked what she was going to do with him, she said she was going to submit him to "faith-healing." After some weeks I went to visit him and found him recovered. His wife confidently attributed his recovery entirely to the "faith-healing." I asked if she had regularly used the sawdust, and she replied most earnestly, "Oh! yes, sir." What her "faith-healing" actually consisted in I did not clearly make out, but she evidently also "kept her powder dry."

This critical and dangerous case was quite as gratifying to me as to the "Faithful" wife of the patient.

III. In private practice in 1906 occurred a case of gangrene beginning in the toe of a diabetic male patient, æt. 62 years, spreading eventually up the leg nearly to the knee. At first I failed to keep down the overpowering smell, even with sublimated wood-wool, which I used after other conventional dressings had been unsuccessful. The perchloridised sawdust immediately stopped all bad odour, not only when covering up the disease, but when the latter was fully exposed along with the incisions in it. As the disease spread I laid open the integuments freely and rubbed the sawdust into the exposed tissues. Here, also, a towel was pinned round the leg, and enclosed plenty of the material, which was changed where saturated—usually once a day. There was no question of amputation in this case, and the patient passed, as expected, into fatal coma. But although no arrest of the gangrene was possible, the room and the diseased leg became sweet, and so continued till the patient's death.

IV. In August, 1909, I was called by a medical friend to his mother, æt. 92 years, who had had an ulcer of the right leg for thirty years. She had been in bed three weeks, and the ulcer was 6 or 7 in. long by 1½ in. wide. It was being dressed frequently without benefit, but she had become delirious, and was supposed to be dying. The only intelligent interference I could suggest was the arrest of any possible septic absorption, in case the delirium was due to that. A towel, containing plenty of the perchloridised sawdust, was pinned round the leg, and examined once a day to remove the small soiled portion. No washing or wiping was done, and the greater portion —that unsoiled—was used as before, slight fresh additions being made over the ulcer. In a couple of days she became conscious, the sore gradually became clean, and after four or five months healed entirely, and she continued as well as her feeble old age permitted. In the last week of 1910 she had a stroke, followed by two others, and died on January 19th, 1911.

As regards II, III, and IV, I do not know of any other dressing or conventional line of treatment by which the desired result could have been attained so easily and cheaply, if at all.

The crude sawdust of white pine should be sifted through a riddle with a one-eighth inch mesh and dealt with as follows: Take of tar oil 2 quarts, hydrarg. perchlor. 120 gr., turmeric 2 oz., water 4 gallons, and mix thoroughly with 4 pecks of the sifted sawdust. Any one can prepare the material for himself, but Messrs. Sumner and Co., wholesale druggists, Hanover Street, Liverpool, keep it in suitable packets ready for use. I find, however, that Messrs. Sumner have been preparing their sawdust from red pine exclusively, under the impression that it absorbs better than the white. I do not believe this at all and have now told them so. Mr. H. O. Thomas and I used the white in the old days, and as it is much more abundant it is easier to obtain. I find also that Mr. Cathcart expresses no preference for the red. Any pine wood sawdust will do, whether mixed or not, and there can be no useful object in selecting a less common variety, and thus increasing the cost. The addition of turmeric gives an agreeable colour to the white pine sawdust, and the tar oil a fragrant odour.

May 24th, 1915.

Gas Phlegmon of the Pia Mater.—Korbsch describes the following case: An infantryman received a tangential shot on the left side of the forehead. He arrived at hospital exhausted and unable to swallow. Highly offensive cerebral tissue was escaping from the wound. A trephine opening showed that fragments of cranium had been driven into the brain. A curette was used to remove the necrotic brain tissue, and the characteristic odour of gas gangrene at once became apparent. After all *débris* had been removed a funnel-shaped cavity remained, the lining of which, still infected, was carefully scraped away. The cavity, which was of the size of a small apple, was packed with iodoform gauze. The patient rallied and seemed out of danger, when fever and mental confusion suddenly developed, and he died soon afterwards. The necropsy showed that the wound was directed towards the anterior horn of the lateral sinus, but had not reached it. The tamponned area seemed to be in a healthy condition. There was leptomeningitis, and the vessels were invested with pus, in which a few bubbles were visible. The condition was at its maximum at the margin of the wound, and gradually became less marked towards the limit of the leptomeningitis on the occiput.—*Med. Record*, vol. lxxxvii, p. 745.

THE FUNCTIONS OF THE OMENTUM.*

By CHARLES F. M. SAINT, M.D., M.S.Dunelm, F.R.C.S.Eng.,
Assistant Surgeon, Hospital for Sick Children, Newcastle-upon-Tyne; Surgical Registrar, Royal Victoria Infirmary, Newcastle-upon-Tyne.

RUTHERFORD MORISON says in his 'Introduction to Surgery': "A special protective agency exists in the abdomen, but for which many abdominal diseases and operations would be attended by a much higher mortality than they are. The omentum, in addition to other uses, may be regarded as the 'abdominal policeman.' It travels about the abdomen with considerable activity, and is attracted, by some sort of information, to neighbourhoods in which mischief is brewing. It may effect a radical cure of hernia, by blocking the hernial orifice with an omental plug. It surrounds, and adheres to, a recently reduced strangulated and damaged loop of intestine, and may keep it alive and prevent a leak. It is generally found in the neighbourhood of a diseased or inflamed appendix; by wrapping it up if gangrenous, or by locking up the pus, from an appendix abscess, it may prevent general peritonitis. In a similar manner it may prevent the perforation of an ulcerating malignant growth or a gastric ulcer, the death of a damaged portion of bowel, or the perforation of a suppurating gall-bladder. When it is found covering and closely enveloping an ovarian cyst, or a fibroid tumour of the uterus, even though not adherent, it is safe to assume that something will be found wrong in the tumour. Its effective mobility is shown by the fact that, whether the lesion be in the diaphragmatic roof of the abdomen, or on the floor of the pelvis, there the omentum can and does find its way.

"Another feature special to the omentum is an extraordinary capacity for the formation of new blood-vessels. Wherever it adheres new blood-vessels develop with great rapidity, and where the continued need for them arises they may reach a considerable size. I have excised large and well-nourished ovarian tumours deprived of their ordinary vascular supply by an attack of torsion,

* Delivered before the Newcastle-upon-Tyne Pathological Club.

and depending for their life on large omental vessels. The only uterine fibroids which can attain to colossal size are either retro-peritoneal, or depend chiefly upon vessels derived from the omentum for their continued growth. The largest I ever removed was of the sub-peritoneal variety. It had a thin and narrow pedicle below, but above was capped by omentum, which carried to it at least half a dozen arteries of the size of the brachial."

From the above remarks the curative influence of the omentum will be readily appreciated, both as to efficiency and diffuse application. A few other considerations are necessary to a fuller comprehension of its beneficial attributes.

We need not delay on such questions as its capacity to keep the viscera warm, in virtue of its fat, its supporting or protective functions, or its office as a store-house of fat, etc.

(1) Absorption of Fluids.

Wilkie ('Brit. Med. Journ.', Oct. 28th, 1911), quoting from his experiments, says: "It was found that removal of the omentum did cause an appreciable, but not a marked, delay in the absorption of saline from the peritoneal cavity, and on the average it was found that the ratio of absorption in animals with intact omentum was to that in animals after excision of the omentum, in the ratio of 3 to 2."

Adams ('Lancet,' March 8th, 1913) quotes Dunmoor, that "in human beings the omentum is capable to taking up 0·5 per cent. of the body weight of fluids per hour, and this absorptive power is proportional to the activity of intestinal peristalsis."

(2) Absorption of Solid Particles.

Wilkie (*loc. cit.*) reports: "The abdomen of a cat was opened, and five grams of sterilised powdered animal charcoal were introduced into the peritoneal cavity and the abdomen closed. In a second cat the omentum was removed and the same quantity of charcoal introduced before closing the wound. The latter animal died two days later, and all the abdominal viscera were found irregularly coated with black powder. The first cat was killed on the second day after operation, and on opening the abdomen it was found that the whole of the charcoal had been taken up by the omentum, and that the other abdominal viscera had a normal glistening appearance."

Adams (*loc. cit.*) reports: "Buxton and Torrey found that almost immediately after the intra-peritoneal injection of inert particles, such as lamp-black, in guinea-pigs, there is a deposit of fibrin on the surface of the omentum in which particles and phagocytic cells of the cavity become entangled, and one hour after the injection the lymphatic channels of the omentum were filled with particles of lamp-black."

Wilkie (*loc. cit.*) further reports: "By injecting a certain dose of emulsion of *Staphylococcus aureus* into the peritoneal cavity in a series of animals, in half of whom the omentum had previously been excised, and then by drawing off the peritoneal fluid, by means of a capillary pipette, at intervals of an hour, one was able to study the cellular changes in the peritoneal exudate in each case.

"It was found that the cellular reaction in the fluid in the animals with no omentum was quite as rapid as, and was even more intense than, in those with an omentum. It was noteworthy, however, that whereas in the latter very few bacteria were visible in the fluid within two or three hours after the injection, in the former cocci were found in large numbers both free in the fluid and within the polymorph leucocytes. By killing one of the animals with an intact omentum at this stage, one could readily demonstrate that the omentum was densely covered by cocci, very many of which were being phagocyted by polymorph leucocytes. The omentum seems to act to germs as a spider's web does to flies. The battle between the bacteria and the leucocytes is transferred from the free fluid to the surface of this vascular peritoneal fold, where the phagocytes are in close touch with their lines of transport and their reinforcements. Little wonder, then, that they stand a better chance of victory."

Allied to these functions is that which is generally recognised, and by virtue of which the omentum encapsules, and so renders innocuous, foreign bodies, such as a sponge or gauze swab, which may have been left in the peritoneal cavity.

The above remarks require no amplification to show what a curative influence the omentum may exert in peritoneal exudates and infections, and also in cases of extravasation of the partially solid contents of the alimentary canal.

Wilkie (*loc. cit.*) has made a special mention of the value of the omentum in infected belly wounds, where it almost invariably becomes adherent, thus strengthening the wall and preventing spread of infection.

Adams also (*loc. cit.*) has demonstrated its utility in covering up raw surfaces by adhesion, probably in many cases only temporary, a fact which is common knowledge in operative work.

The aptitude with which the omentum prolapses and so plugs a perforating wound of the belly wall resembles somewhat the plugging of perforations of the gut, etc. (*v. s.*).

Another common observation, made at operations, is the remarkably rapid and effective formation of adhesions to mops, instruments, etc. So perfect may these become during the course of the operation that oozing of blood may attend their separation. Another example of the same characteristic is the remarkable way in which a tag of the omentum will persistently adhere to and follow the withdrawal of the closed ends of a pair of scissors, at the closure of the last small portion of peritoneal wound.

The greatest controversy has perhaps waged round the question of the "active" or "passive" nature of the movements of the omentum. Of the men who have written on this subject, Rutherford Morison is perhaps the strongest upholder of the "active" hypothesis.

Let us first see what case the opposition presents, and then consider what may be advanced against it.

In favour of the passive and non-selective nature of the movements of the great omentum numerous experimental and other observations have been adduced, some of which are as follows:

(1) Adams (*loc. cit.*) fixed two lead shot in the right and left free borders of the omentum of a rabbit and then closed the abdomen. Three months later, the rabbit, being in good health, was X-rayed every half hour for six hours, and it was found that the range of movements was not great. From this it was deduced that "it appeared to be absolutely certain that the omentum was entirely dependent for its movements on the peristaltic waves of the bowel; further than this, that its movements were slow, and that inflamed or injured areas of peritoneum, though they often arrest the omentum till it becomes adherent, do not attract it by any special chemiotactic or physical influence."

(2) Wilkie (*loc. cit.*) quotes the following experiment as conclusive:

"Under ether anæsthesia the abdomen of a rabbit was opened. Its omentum was lifted up and laid over the liver. The left hand corner of the omentum was fixed with one catgut stitch to the parietal peritoneum in front of the liver. An emulsion of *Staphylococcus aureus* was then injected into the submucous and subperitoneal coats of the appendix. The animal died five days later from appendicitis with localised peritonitis. Post-mortem it was demonstrated that the omentum had not made the slightest movement in the direction of the inflamed appendix, although its right-hand border could readily be lifted down to the seat of trouble." The author proceeds:

"This was no case of negative chemiotaxis, merely the inability for combined action between those two physical factors which determine the position taken up by the omentum—viz., respiratory and peristaltic movements and peritoneal irritation and roughening."

"The movement of the omentum to an infective zone is not a matter of minutes, but of hours or days; consequently one practically never finds the omentum plugging a sudden perforation of a hollow viscus, *e.g.* a traumatic rupture of the intestine. The fact noted by Durham, that in a rapidly fatal case of general peritonitis the omentum is found curled up under the stomach, I have repeatedly observed, and agree with him that this position is determined by peristaltic movements and not by negative chemiotaxis. Repeated examinations have failed to discover any evidence of muscle fibres in the omentum."

"Inflammatory processes which are situated outside the normal range of the omental excursions do not attract that organ, *e.g.*, an inflammatory focus at the bottom of the pelvis is usually entirely ignored by the omentum, unless the latter happens to be unusually long and pendulous."

(3) Adams, again, quotes: "One case is of especial interest because at the same operation the serous coat was detached from a portion of the colon and the lower half of the omentum was removed; the omental stump did become adherent to the colon, but about one inch from this bare area, which was itself free from all adhesions, showing no evidence of inflammatory repair. This animal was killed after only twenty-six hours, but

the result suggests the absence of selective power on the part of omentum, since the bare area was easily within its reach."

Let us examine these observations in order.

(1) This experiment appears to me to justify none of the deductions made from it, but rather to provide an interesting study of the range of movement of the omentum in a normal abdomen, *i. e.*, when no pathological process is present. We have further to consider that the movements may have been incommoded by the weight of the shot.

(2) First let us consider the experiment quoted: "The animal died five days later from appendicitis with localised peritonitis." How far are we justified in accepting that this was the whole of the pathology? No record is given of a blood examination, and one must suppose none was made, so that the actual cause of death may have been an accompanying septicæmia. In such a case, presupposing an actively mobile omentum, it does not require much imagination to suppose that the animal is so overcome with the infection that little or no local response is found; hence the inactivity of the omentum. I shall have more to say of this later. On the other hand, we must keep in mind that the omentum was tacked down and hence was acting under abnormal conditions. It is too much to assume that Nature provides a normal activity under such circumstances, and therefore the experiment is not analogous to a true ordinary case of appendicitis. One might mention here the common clinical observation that gut which is reduced into the abdomen, after strangulation in a hernial sac, remains at the site of its reduction, *i. e.*, it does not move about. This is, perhaps, not strictly analogous to the omentum which has been interfered with and fixed down. If, however, the omental movement be entirely passive, and dependent on peristalsis, there is no adequate explanation of its failure to become adherent.

In the light of these remarks, the statement which follows the description of the experiment appears to be purely speculative and to be based on hypothetical grounds.

The next point is the question of the time taken for the omental movements—considered to be slow by both Wilkie and Adams. Adams certainly quotes nothing in his paper to substantiate his view, and Wilkie brings forward only his own clinical experiences, viz., "consequently one never finds the omentum plugging a sudden perforation of a hollow viscus." Even in Wilkie's experience it is just "practically never," so that in some of his cases it *was* found plugging a sudden perforation, and therefore he grants that its movements are *sometimes* rapid. Perhaps other authorities would be prepared to give a higher proportion of these cases, plugged with omentum. Certainly nothing could be much more rapid than the speed with which a prolapse of omentum will take place in a perforating wound of the belly wall, so plugging it.

The next fact to be dealt with is the observation "that in a rapidly fatal case of general peritonitis the omentum is found curled up under the stomach." Surely the comment that "this position is determined by peristaltic movements and not by negative chemiotaxis" is a misinterpretation of the incident. Rather, I think, one might look for analogy in other reactions on the part of the body to similar rapidly fatal infections. Thus, in these cases where a wound is infected and a rapidly fatal septicæmia develops, the local inflammatory reaction is often almost absent, the body being apparently overwhelmed by the general infection. Again, while in the ordinary course of events we look for leucocytosis as one response, on the part of the blood, to infection by pyogenic organisms, we know that in the most severe infections no such development takes place, the individual being apparently overcome. Similarly, in acute infections, while the body may respond in the early stages by the occurrence of fever, in the worst cases this rapidly disappears and the temperature may become subnormal. In all of these cases, we see that the ordinary reaction of the body does not take place, and it seems probable that the omental incompetence may be similarly explained. One might even go further than this and, instead of looking on this "curling up" of the omentum as the result of passivity, regard it as an active recession from the field of a hopeless contest. This apparent aversion is best seen in cancerous growths. It is worth keeping in mind, too, that in these rapidly fatal cases of general peritonitis peristalsis is entirely absent, the gut being paralysed, and so the part played by peristalsis must be limited or negative.

The next statement which calls for remark is that of Wilkie, viz., "Inflammatory processes which are situated outside the normal range of the

omental excursions do not attract the organ"; and he cites a focus at the bottom of the pelvis. The first question that arises is, "What is the normal range of the omental excursions?" On this subject, Wilkie, in the paper quoted, gives no enlightenment, and all we can bring to our help is Adams' experiment (*v.s.*) in which he states that "the range of movement was not great." It is important to remember that Wilkie modifies his own statement, in so far as he says, "An inflammatory focus at the bottom of the pelvis is *usually* entirely ignored by the omentum, *unless the latter happens to be unusually long and pendulous.*" In other words, he says that where the omentum is able to reach the inflammatory focus, it does so.

The following case appears to be of considerable importance in the question of the active or passive nature of the omental movement. M. S—, female, was under the care of Professor Rutherford Morison at the Royal Victoria Infirmary, February 1st, 1913. On opening the abdomen, the liver was found to be occupied by a large suppurating hydatid cyst, which had come to the surface on its diaphragmatic aspect. At this spot the omentum was widely adherent, *i.e.* between the liver and diaphragm.

Another type of case, important in this connection, is that of ovarian tumours. In an uncomplicated ovarian tumour the position of the omentum is along its upper border, so that it presents a crescentic arrangement with concavity downwards. When, however, as I have quoted above, "something is wrong with the tumour," the omentum wraps it up as far as possible, and so comes to lie on its anterior surface, between it and the anterior abdominal wall. In order to attain this position, it must, as it were, "climb over" the dome-like elevation of the tumour.

The special significance of these cases is the fact that the omentum was adherent or, at any rate, present at a site removed from the sphere of peristaltic movement, so that some other factor than this must be presupposed, the most likely being the spontaneous active movement of the omentum.

The experiments quoted above, in which inert particles were introduced into the peritoneal cavity, and also organisms, appear to me to corroborate this view. We have seen that, when the omentum is present, both inert particles and organisms rapidly disappear from the free peritoneal fluid and are found on the omentum, whereas, if the omentum has been removed, they remain distributed throughout the cavity, free in the fluid exudate. Those who vouch for the passive nature of the omental movements necessarily preclude the possibility of any chemiotactic influences, and so attribute all to the peristaltic and respiratory movements. Here we find certain inconsistencies. Both Wilkie and Adams emphasise the slow rate of omental movement and the limitation of its range, yet we find that Wilkie states that within two or three hours very few bacteria are visible in the peritoneal fluid, and, in the case of lamp-black, "the other abdominal viscera had a normal glistening appearance"—evidently in this scavenging process the pelvis is no exception to the rest of the peritoneal cavity. A point of the greatest importance in the injection of organisms is that peritonitis entails diminished or absent peristalsis, which is Nature's method of inducing rest in the part and controlling the spread of the infection. Under these circumstances, one would reasonably have expected that the omental movements would be diminished, rather than increased, as would appear to be the case from the above considerations, and as quoted by Wilkie in another place.

(*To be concluded.*)

May 24th, 1915.

Death following Intraspinal Injection of Novocain.—Scandola's patient was a priest, æt. 69 years, with signs of a mild myocarditis, so that when a herniotomy was undertaken the lumbar route for anæsthesia was chosen rather than that by inhalation, and only a small amount of novocain solution was injected. The patient bore the operation well, and seemed to be in the best of condition that night, but next day there was some delirium, and signs of paresis of the intestines gradually became evident, with death on the third day. The delirium had continued all the time. The necropsy showed no signs of peritonitis. Merusel reported last year the death of two patients of 75 and 80 after intraspinal injection of a small dose of novocain. In several other elderly patients there was sub-delirium, requiring constant surveillance, but the patients recovered after four or five days. Scandola has also had collapse in an elderly patient, who recovered finally under stimulants. The spinal nervous system seems from these and other data cited to be peculiarly susceptible in the elderly.—*Journ. A.M.A.*, vol. lxiv, p. 866.

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE ON A CASE OF ANTHRAX.*

By W. SAMPSON HANDLEY, M.S., F.R.C.S.,
Surgeon to the Hospital with care of Out-Patients,
Captain R.A.M.C.(T.F.)

WITH REMARKS ON THE PATHOLOGY OF THE DISEASE.

By C. H. BROWNING, M.D.,
Director of the Bland-Sutton Pathological Institute.

GENTLEMEN,—The age of the patient whom you see before you is thirty-eight, and she is a brush-maker. A fortnight ago she noticed a small red speck just below the left eye. She did not attempt to squeeze or prick it, but applied boracic fomentations—a very sensible treatment. As the spot rapidly got larger, and became very painful, she went to Dr. Long, of Chesham, on the Thursday evening. There was then considerable swelling on the left side of the face and neck, and some discoloration. Dr. Long did not arrive at a diagnosis at once, but he prescribed an ointment, and told the patient to call again next morning. She could not sleep owing to the pain, and she saw the doctor twice the next day. The swelling and pain were intense. That is not the rule in anthrax, the pain is not usually severe. On Saturday the patient was admitted to the Middlesex Hospital, Dr. Long having sent her with a diagnosis ready made. The disease had then been developing from the Tuesday to the Saturday—four days.

On admission, her pulse-rate was 80, her respirations 20, and temperature 98·6° F. I show you the temperature chart of the case, from which you will see that on admission the temperature was practically normal, but very shortly after admission it rose to something over 99° F. I was asked by Mr.

* Delivered at the Middlesex Hospital.

Lloyd Williams to see the case. On the left side of the face, in the situation of the gap which you see, there was an indurated mass, raised above the general level, mottled with patches of black, and presenting here and there on its surface vesicles filled with some purulent fluid. That is not the typical picture of an anthrax pustule. However, we took some of the fluid from the vesicles, and made smears, which were examined by Dr. Browning, Director of our new "Bland-Sutton" Institute of Pathology, who found the anthrax bacillus. We tried to obtain some Sclavo's serum, but, as far as we could ascertain, none was to be obtained in London. So I took the patient into the theatre and excised the mass, leaving the gap which you observe; it is now a healthy granulating wound, and there is nothing characteristic of anthrax about it, except that on the lower eyelid you will notice a black slough. That is situated at a point where I did not excise the lump so widely as round the rest of the circumference, because I could not do so without spoiling the lower eyelid, and I could not bring myself to remove a portion of that. That black slough gives the characteristic appearance of the central part of a typical anthrax pustule, although in this case the active symptoms have passed. I also tied the facial vein and the angular vein, so as to shut off channels by which the anthrax bacilli might reach the brain, or pass down to the lungs, and set up septicæmia or broncho-pneumonia. After the operation was finished, I injected 1 in 20 carbolic in the region round the place where the malignant pustule had been, using about two drachms of the fluid. The wound was dressed with a pad soaked in crystal violet, a dye which, Dr. Browning tells me, is very poisonous to the anthrax bacillus. The next day the swelling had increased, and had extended to the opposite eyelid and to the opposite side of the face; the temperature rose to 101° F. On November 23rd she was decidedly better, and on the following day the temperature began to fall, reaching the normal on the 25th. On that day she was practically well, and she has remained so since. I intend soon to skin-graft the granulating wound, and I do not think she will be very much disfigured, after all.*

* This prophecy proved to be correct. Nevertheless, upon her discharge the patient observed that "it was a good job she was married."

Now for some general considerations on the subject of anthrax. As you all know, the disease is due to inoculation with the bacillus anthracis, and it is essentially a veterinary disease. It is very prevalent in some foreign countries, and even in France it causes great loss to the farmers. I will read you an extract from the 'Life of Pasteur,' by Vallery Radot, which gives you a good idea of its incidence in France: "Some farms were called *charbon* farms"—charbon is the French name for anthrax—"elsewhere, certain fields or hills were looked upon as accursed, and an evil spell seemed to be thrown over flocks bold enough to enter those fields or ascend those hills. Animals stricken with this disease almost always died in a few hours; sheep were seen to lag behind the flock, with drooping of the head, shaking of the limbs, and gasping breath. After a rigor and some sanguinolent evacuations, occurring also through the mouth and nostrils, death supervened, often before the shepherd had had time to notice the attack. The carcase rapidly became distended, and the least rent in the skin gave issue to a flow of black, thick and viscid blood; hence the name of anthrax given to the disease." It was also called splenic fever, because necropsy showed that the spleen assumed enormous dimensions. If the spleen were opened it presented a black and liquid appearance.

There is another interest attached to the disease in animals, because it is one of the first diseases to which protective inoculation was successfully applied. In a crucial experiment Pasteur took 20 sheep, 10 of which he had protected beforehand by his method of inoculation. All 20 sheep, previously inoculated with the deadly anthrax virus, were placed on public view at a great agricultural show in France. The ten protected sheep survived, while those which were not protected all died. And Pasteur tells with what anxiety he waited for the result, alone among a crowd of sceptics. The scene is reminiscent of that great controlled experiment on the power of Deity related in the Old Testament, an experiment curiously modern and scientific in its conception. You remember Elijah's challenge to the priests of Baal, the rival sacrifices, the rival prayers to Heaven for fire to descend and consume them. At the end of the day in his case, as in Pasteur's, the energy of an individual triumphed over the unbelief of the crowd, and those inseparable companions, faith

and work, were justified. I suspect that as religion was the consolation of Pasteur's age, science was the relaxation of Elijah's leisure moments. His methods betray the scientific mind.

Anthrax in man is an occupation disease, occurring in those people who come into contact with hides, skins, or the wool and hair of animals. Consequently it does not occur much in women; only about 2 per cent. of the cases are in women. Its distribution, geographically, is very local. Thus in London it occurs in Bermondsey and Rotherhithe, districts where tanning is the chief occupation. As many as a hundred cases could be collected from the records of Guy's Hospital, which serves those districts, while the other hospitals in London have only an occasional isolated case.

There are four forms of anthrax in man, but only two of them need detain us. The disease may be divided into external anthrax and internal anthrax. And there are two varieties of external anthrax, known as malignant pustule and malignant œdema.* There are also two varieties of internal anthrax, known as wool-sorter's disease and intestinal anthrax.

It is only the local varieties which need now concern us. Malignant œdema, in which the local lesion is absent while there is a widespread œdema of the limb, is very rare; so we are reduced to the consideration of malignant pustule. The site is usually on an uncovered part of the skin from inoculation of the bacteria during the handling of raw hides, more rarely of bristles. Malignant pustule occurs most frequently on the face or neck. It appears first as a pimple simulating an insect bite, which causes local irritation, but not much pain. Advice is usually sought by the patient on the third day, because by that time the œdema of the surrounding tissues is beginning, and on the third day the typical condition has developed. In the centre there is a black slough, which is surrounded by a ring of little vesicles, and that, again, by an area of redness and œdema, the œdema extending beyond the redness. Remember, then, those three zones in the anthrax pustule. I show you an early coloured illustration in a book. The tissues of the central area are stained black by extravasated and altered blood. The fluid in the vesicles usually contains abundant bacilli. Even if you take away nothing else from this lecture, I want you to particularly remember these three zones.

The constitutional symptoms are not usually severe, but there is headache, dizziness, and occasional vomiting and rigors. The lymphatic glands are sometimes enlarged, but not always. In this present case they were not enlarged; and it is probable when they are not enlarged that it is a good element in the prognosis. In cases which are going to be fatal, the œdema extends, and the whole head, for instance, may be enormously swollen; the pulse rises to 120 or so and the temperature goes up to, perhaps, 104° F.; there is vomiting, diarrhœa, restlessness, and these symptoms are followed by delirium, coma, and death within a week of the onset. I now show you the piece removed from the patient you saw. You will see the blackened areas which mottled the pustule, and that the vesicles are filled with semi-purulent fluid.

A few words are necessary on the diagnosis of anthrax. Any local septic lesion in a patient working amid hides, skins, or bristles should arouse suspicion. If the suspicion of anthrax is aroused in your minds it is easy to settle the question. You should make smears of the serum from any vesicles which are present in the lesion, and either examine them yourself by Gram's method, or send them to a laboratory for examination. The great thing is to think of the possibility of the disease; for if the idea occurs it is easy to settle whether anthrax is present or not.

The conditions which most closely simulate anthrax are, firstly, a septic mosquito or gnat bite, in which there is an indurated mass under the skin. But in these, of course, there is an absence of the characteristic features of the anthrax pustule. The other condition which closely resembles in appearance anthrax is carbuncle. In that, again, there is a subcutaneous induration; generally it is on the face or the back of the neck, but there is no central black slough, and there are no vesicles. In the carbuncle pain will be much more acute, and suppuration will probably be visible on its surface moreover, when you press on the centre of the mass you will feel a softening, which indicates commencing suppuration. There is none of that softening in the centre in an anthrax pustule. There is one other condition which was said by the late Mr. Davies-Colley to simulate anthrax, and

* The term "malignant œdema" is ambiguous, since it has been also applied to the "gas gangrene" resulting from infection with certain anaerobic bacteria.

that is primary chancre of the face. But in the latter case the development of the lesion will be much slower than in anthrax, the glands will soon become enlarged and will present the characters of secondary glands, and so I cannot think you will have much difficulty in distinguishing them.

Now a word about the outlook, the prognosis, in anthrax. The mortality, among properly treated cases, is only 9 per cent., and recovery is possible even when the bacilli are present in the circulating blood. That is an interesting fact, which is shown by some of the recorded cases. It is also fairly certain that some cases recover even when they are not treated. But if a case be not treated death is the usual result.

With regard to the treatment of the disease, broadly speaking, there are two methods. You can excise the malignant pustule freely, injecting some antiseptic into the tissues around. Or, in the second place, you can trust to the injection of a protective serum, which is called Sclavo's serum. The treatment usually adopted in this country is that of free excision, swabbing the raw surface left with pure phenol and injecting 1 in 20 carbolic into the tissues around by means of a hypodermic needle. The excision should be carried down as far as the deep fascia. Mr. Davies-Colley, who had great experience of anthrax, recommended dressing the wound with powdered ipecacuanha; and Muspratt, who introduced that treatment, thus treated fifty cases without a death. But the method I advise you to adopt is free excision, with injection of 1 in 20 carbolic into the tissues around.

Now we must consider the second method—treatment by serum. This serum is obtained from an ass which has been protected against anthrax by the injection into it first of a very old and weak culture of anthrax, later by a young culture, and finally by a succession of virulent cultures in increasing doses. By this means the blood of the animal acquires protective properties similar to those possessed against diphtheria by the blood of a horse treated with cultures of that disease. The initial dose of Sclavo's serum is 40 to 50 c.c.; but much larger doses may, if desired, be given. The injection is followed by a rise in temperature, to about 105° F.; but this rise is associated with improvement in the symptoms.

With regard to the efficiency of the serum treatment, I think the best piece of evidence I have come across is that in a certain town in Italy, where anthrax is rife, the workpeople now refuse any other treatment except the serum treatment. I imagine they are fairly good judges, since they must see the results of that treatment in a number of cases. In this country the difficulty of obtaining the serum is a serious obstacle. If you see a case of anthrax early, it seems to me right that you should endeavour to obtain the serum, and if you can get it, to trust to the use of the serum without excising the pustule. That is particularly the case if the pustule is situated in a part where excision would produce great deformity; for instance, on the eyelid. But if you do not see the case until late, when already there is extensive œdema, or if you are unable to obtain a supply of the serum you should freely excise the pustule, without much regard to æsthetic considerations.

You will have noticed that I have not spoken at all on the bacteriology of anthrax. The reason of that omission is that I have asked Dr. Browning—and he has consented—to come and give you a brief description of the bacteriology of the disease. There are, however, two points I would like to insist upon, because they bear upon the clinical characters of the disease. The first is, that inside the body anthrax does not produce spores. The second is, that outside the body anthrax does not produce a toxin; bacteriologists have never been able to obtain the anthrax toxin from cultivations of the anthrax bacillus in a test-tube.

You may possibly think that, in directing your attention to such a rare disease, I have been practically wasting your time. Many of you will go through your professional careers without ever seeing a case of anthrax. But others, sooner or later, will meet with the disease; and the recognition and proper treatment of the case will make all the difference between the life and the death of the patient. My object in giving this lecture has been to try to ensure that in future anthrax shall occupy a retired, but none the less a permanent, corner in your minds; then you will not be likely to miss any case which happens to cross your path.

I would like to say, in conclusion, that the patient you have just seen owes her life to an old Middlesex Hospital man, who had preserved, through years of routine practice, the alert and

open mind, which enabled him to recognise the disease.

Remarks by Dr. Browning.

The absolute diagnosis of anthrax, like the absolute diagnosis of diphtheria, depends on obtaining the specific organism from the patient. The anthrax bacillus may be obtained from a malignant pustule comparatively readily, as was the case here. But sometimes the bacilli are very scanty, and a culture may yield a positive result where the direct microscopic examination of the exudate is negative. Again, it may not be possible to recover the bacilli, in which case, of course, the diagnosis is only presumptive. If you make a film of the exudate in the pustule and find organisms with the characteristic appearance, lying singly, or in chains, you can be practically certain you are dealing with *B. anthracis*. They are Gram-positive, oblong bacilli, with rather square ends, bamboo-like, as has been said, and they grow well on ordinary culture media, such as agar-agar.

By staining with watery methylene-blue, a characteristic appearance is obtained; the bacilli are seen to be surrounded by a capsule, which appears as a distinctly violet halo (M'Fadyean's reaction). So characteristic is this appearance that if you found such capsulated bacteria in material from a case, you could be certain you were dealing with *B. anthracis*. You would never get organisms of this kind from a human lesion unless the case was one of anthrax. But, of course, the evidence is completed by obtaining a culture, and then proving that the organism is pathogenic —*i. e.* that it causes septicæmia in a susceptible animal, such as a mouse or a guinea-pig, which are the two most susceptible animals. In the cases of an organism, with the appearances above described, from a human lesion, the result of the animal experiment should not be waited for in arriving at the diagnosis. The microscopic examination of smears and cultures will not suffice, however, if you are examining the material which is suspected of causing the infection—hides, or, as in this case, hair used in brush-making. Here the problem is more complicated since, in the outer world, you may quite well meet with organisms which look like anthrax in culture and under the microscope. Such "anthracoid" organisms may present the closest resemblance to *B. anthracis*, but they are not pathogenic. So that, to clinch the diagnosis, in tracing the infecting agent to material, such as hair or hides, you must also prove that the organism obtained in culture is pathogenic.

As Mr. Handley has already impressed on you, spores are not formed in the tissues by the anthrax bacillus; this is very important, because it means that you can readily deal with the infected material and render it non-infective. But when material containing anthrax bacilli has stood in contact with the air, spores are produced. When spores have been devoloped, the material is extremely dangerous from the hygienic point of view, because it is so very difficult to disinfect it. This has a direct application in dealing with the carcases of animals, for, if an ox has died of anthrax, the intact carcase can be comparatively readily rendered innocuous, because the bacilli are in the non-spored condition, and are susceptible to ordinary antiseptics. But when the carcase has been opened up and the tissues exposed to the air, it becomes very difficult to disinfect, since spores form, which will withstand exposure to ordinary antiseptic solutions for many hours or even more than a day. It requires boiling for five or ten minutes to sterilise material infected with anthrax spores.

There is an interesting fact connected with the pathology of anthrax which is not as well emphasised as it might be, that sometimes one meets with cases of obscure origin which, on post-mortem examination, show hæmorrhagic lepto-meningitis. I am speaking of cases where no question of trauma has arisen; where the person has died from an obscure illness, and post-mortem practically all one may find is an exudation of blood on the surface of the brain. In such cases the suspicion of anthrax infection should always be entertained. And, of course, bacteriological examination should be made, as considerable harm might be done by failing to recognise the nature of the condition.

May 31st, 1915.

THE FUNCTIONS OF THE OMENTUM.

By CHARLES F. M. SAINT, M.D., M.S.Dunelm, F.R.C.S.Eng.,

Assistant Surgeon, Hospital for Sick Children, Newcastle-upon-Tyne; Surgical Registrar, Royal Victoria Infirmary, Newcastle-upon-Tyne.

(*Concluded from p.* 176.)

THE last point which requires comment is the "non-selective" nature of the omental movement.

In Adams' experiment (*v.s.*) half of the omentum was removed and the colon denuded of its peritoneal coat, the stump becoming adherent to the colon, about one inch from the denuded surface. The special feature of this experiment, which appears to me to render invalid the deductions drawn, is the fact that we are dealing with a grossly abnormal omentum, one in which the lower half, *i. e.* the free border, was removed at the time the denuded surface was made. Under these circumstances, could we reasonably expect the omentum to behave normally, and is Adams justified in assuming that the adhesion which occurred to the colon was some abortive effort to cover the bare surface? One is almost inclined to strain analogy, and say that those who would expect the omentum to act normally under such conditions as the above, would expect an upper limb, after amputation, through the forearm, to functionate the same as before amputation, *i. e.* when hand and fingers were intact.

On the other hand, in addition to all the evidence of a selective power on the part of the omentum, given above in the passage quoted from Rutherford Morison, the following case may be cited. The patient, a female, was operated upon by Mr. W. G. Richardson at the Royal Victoria Infirmary. A ruptured ectopic gestation was found, the fœtus lying free in the peritoneal cavity. No adhesion of any kind had taken place except that the omentum was found tightly bound to the perinæum, completely enclosing the anus, scrotum, and penis, so much so that the fœtus could be lifted up by its omental pedicle. By this adhesion, the anile and penile orifices were effectually sealed and fouling of the peritoneum prevented. In such a case, the deduction of a positive selective power on the part of the omentum seems incontrovertible, and the onus of disproof rests with the opposition.

Assuming the passive nature of the omental movement, some other possibilities have been suggested, for which I am indebted to Dr. Menzies. Briefly, they are:

(1) The presence of a fluid wave carrying the omentum to the seat of trouble. Against this we have the fact that the omentum absorbs rather than secretes fluid, and hence the wave would tend to be away from the focus. In addition to this, there is little doubt that the most of the fluid secreted arises at the site of irritation.

(2) The absence of movement at the seat of trouble may lead to the arrest of the omentum at that spot, like the eddies produced in the marginal recesses of a stream, the peristalsis acting as the motive force. If this were correct, one should expect that in all cases with localised rigidity of the abdominal wall or paresis of the intestine the omentum would be found in that situation. This is by no means always so. On the other hand, where general peritonitis is present, with general rigidity and intestinal paresis, we should not expect the omentum to become adherent at the primary focus. This, however, it not uncommonly does. Again, this hypothesis does not explain the omentum becoming adherent in areas outside the range of peristaltic movements—*e. g.* the diaphragmatic surface of the liver or the front of a large ovarian cyst—nor can it explain an adhesion to the posterior surface of the mesentery by turning round the free border of the gut. Lastly, as I have mentioned above, in the experiment where organisms were injected into the peritoneal cavity with the production of peritonitis, the range of movement of the omentum would be expected to be restricted, and, as a consequence, the absorption of the organisms to be prevented—*i. e.* from absence of contact. In the case of the injection of inert particles no rigidity or paresis results, and yet the particles become absorbed.

We must next consider the question of the possibility of spontaneous movement of the omentum, or whether it is entirely dependent, as the above-mentioned and other observers vouch, on the peristaltic and respiratory movements. The great stumbling-block to the acceptance of its spontaneous movement has been the statement that, as Wilkie observes, "repeated examinations

have failed to discover any evidence of muscle-fibres in the omentum." This may be correct for the general fatty ground-work of the omentum, but, as Mr. W. G. Richardson has suggested, the omentum is singularly well off for blood-vessels, and the middle coat of these is mostly composed of muscular tissue. He has advanced the hypothesis that spontaneous movement of the omentum takes place in virtue of this vascular muscle-tissue, the stimulus being in most cases chemiotactic. The necessary mechanism for active movement is thus provided, and the factor which has hitherto been lacking is supplied. The hypothesis awaits substantiation.

Apart from this possibility, we must keep in mind that the power of active movement is not limited to muscle-tissue. In the field of zoology this is commonplace—*i. e.* among the lowest forms of life, *e. g.* amœba, etc. In the human body it is typified in the leucocytes. It must be remembered that the omentum is the posterior mesogastrium, and as such comprises similar mesodermal tissue to that which gives rise to the muscular walls of the alimentary canal, and hence it is just the tissue which one might expect to be possessed of amœboid movement. Certainly, too, in the mesentery of the newt, a lower portion of the posterior mesentery of the alimentary canal, striped muscle-fibre has been demonstrated. Should this hypothesis be correct, then doubtless the stimulus would be for the most part chemiotactic, though doubtless peristaltic and respiratory movements would be an important accessory. However that may be, like the last-mentioned hypothesis, it is for the moment *sub judice.*

Albrecht has described the omentum as a "large abdominal leucocyte," and I venture to suggest that, in addition to its other functional activities, he had in mind a possible analogy to the pseudopodia.

Other pathologists prefer to think of it as a "brooding providence," and one cannot but feel convinced that, in spite of the absence of positive proof, such a designation was the expression of the belief, in the mind of the inventor, of its active and selective movement; for the very term means a "watchful foresight," and involves that which takes place as a result of it. It would, therefore, be utterly wrong to apply it to any passive, non-selective agent.

May 24th, 1915.

Subacromial Bursitis.—S. O. Fields points out that not until Codman's paper, in 1906, was much interest manifested in subacromial bursitis. Previously the symptoms were ascribed to "rheumatism," contusion of the deltoid muscle or of the bony structures in the vicinity, sprains of the shoulder-joint, and what not. Of late, however, we have grown to appreciate the *rôle* of lesions of the bursa in the production of the greater proportion of stiff, painful, and disabled shoulders. But the lesion, with its unmistakable signs and symptoms, and its untoward results, is often overlooked by the practitioner, simply because he does not bear it in mind.

The subacromial bursa lies beneath the acromion process, being interposed between the major tuberosity of the humerus and the tendinous expansion of the supraspinatus below, and the deltoid, the coraco-acromial ligament, and the acromial process above. Formerly it was believed that there existed two separate bursæ—the subdeltoid and the subacromial—but we have learned that the so-called subdeltoid bursa is merely an extension of the subacromial. The sac is a modified connective-tissue space lined by an imperfect cellular layer, and contains a small quantity of fluid for lubricating. The sac exercises a ball-bearing function, especially in motions of the shoulder involving abduction and rotation.

Text-books say that trauma, especially of the shoulder directly, is the chief cause. But, excepting that portion intervening between the deltoid and the major tuberosity, the bursa is well protected from direct injury of the shoulder, and one sufficient to produce an inflammatory lesion would of necessity cause tremendous damage to surrounding structures. Falls have decidedly a greater ætiological *rôle*; but not falls on the shoulder, as so commonly stated, for a fall on the shoulder has been shown to be well-nigh impossible. Consciously or unconsciously, the hand or elbow on the threatened side is thrown out to catch the force of the impact. It follows that the force resulting from the fall travels up the abducted arm and damages the bursa between the major tuberosity of the humerus and the acromion.

In the cases of subacromial bursitis coming under the writer's observation, chiefly in labourers, the proportion of blows upon the shoulder was remarkably small, that of falls upon the shoulder practi-

cally *nil*, and the proportion of falls upon the elbow and outstretched hand greatest of all. The factor of prolonged and excessive use of the shoulder by untrained and inexperienced men, especially in abduction and rotation, implying fibrillary tears of the tendon of the supraspinatus and consequent involvement of the bursa, which is intimately associated with the tendon of that muscle, should not be forgotton.

In the text-books slight emphasis is laid on infection. There is at most a mention of sepsis, gonorrhœa, etc., as occasional causes. The writer had the importance of infection in subacromial bursitis forced upon him by observing the not inconsiderable proportion of cases of the affection which followed newly contracted gonorrhœa, or the flaring up of an old "cured" or latent gonorrhœa. In these cases the shoulder gave no trouble until the patient had worked himself to the point of exhaustion, contracted a cold from becoming chilled at the dinner-hour, or indulged in alcohol about three weeks after infection. In more than one case a history of sensation of chilliness, general malaise, and feverishness, preceding the shoulder pain by a few hours, could be elicited. In two other cases the bursal symptoms developed about two and a half weeks after severe attacks of tonsillitis, suggesting the possibility that the tonsils are capable of playing the same *rôle* in the production of bursitis as in the production of arthritis. In none of these cases of infectious bursal inflammation was there a history of any shoulder trauma, either direct or indirect. Thus there is an apparent analogy between metastic arthritis and the infectious bursitides.

Subacromial bursitis is frequently overlooked from want of care. Pain, tenderness, and limited disability in the shoulder region succeeding trauma, or following after an interval of two or three weeks a mucous membrane infection, are the cardinal symptoms. At first the pain is usually intense, even the mere weight of the unsupported arm causing intolerable suffering. The painful area may be circumscribed, but occasionally there is wide diffusion of the pain, even as far down as the wrist. Any attempt at abduction or rotation increases the pain.

Limited disability, due in the early stages to the protective muscular spasm and the painful friction between the upper and lower walls of the bursal sac, is characteristic. The arm can be abducted, passively, and at times actively, to the extent of 12° or 15° before muscular spasm locks the shoulder-joint, thus compelling the performance of further abduction by rotation of the scapula on the thorax. Only the slightest external and internal rotation, or none at all, is possible. Later, especially in untreated or badly treated cases, the disability is due to adhesions between the bursal walls or to a thickened sac wall.

A conspicuous feature is tenderness involving in most instances the entire shoulder with usually an area of maximum intensity over the extreme upper and outer point of the shoulder just external to the acromion. Other occasional signs are palpable friction rub and enlargement of the bursa, causing increased prominence of the shoulder.

The prognosis is nearly always good; willingness to use the shoulder after subsidence of the acuter manifestations helps recovery.

The treatment recommended consists in retention of the shoulder in abduction, mild counter irritation, cautious massage, and later, gentle but positive and active motion. Obliteration of the bursal sac by the injection of irritants or its excision may be practised as a last resort. Dissatisfied with the somewhat lengthy period of disability under the foregoing treatment, the writer tried a method used in arthritis—aspiration of the inflammatory secretion and injection of some agent to neutralise further inflammatory secretion and to stimulate leucocytosis—2 to 4 c.c. of 5 per cent. iodoform in glycerine. The forearm is slung across the chest, the hand being kept as near as possible to the opposite shoulder. Relief of the intense pain is almost immediate. After twelve days gentle massage of the shoulder region and gradual passive and active motions are performed, returning the arm to the sling after each exercise. Usually in three days more the sling is permanently removed and the patient encouraged to move the shoulder freely.—*New York Med. Journ.*, p. 163.

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We regret that owing to the war the number of pages must be diminished for a time.

AFFECTIONS OF THE EYE AND ORBIT IN DISEASES OF THE NASAL ACCESSORY SINUSES.

By D. LEIGHTON DAVIES, M.S.Lond., F.R.C.S.,

Assistant Lecturer on Anatomy, University College, South Wales; Assistant Ophthalmic Surgeon, Cardiff Infirmary.

THERE is no organ in the body the function and well-being of which are not affected in some degree by diseases of or changes in neighbouring or distal structures or organs. This can occur in two ways: (1) By direct involvement in the case of adjoining organs where one is diseased, or (2) in a more subtle manner, through the blood-stream. In connection with this we must bear in mind that every organ or tissue which, as a result of some disorder or disease, including inflammatory affections, does not functionate normally, is taking away some substance or substances from the blood, or adding others which are not usually to be found in it; this apart altogether from the presence of micro-organisms as we at present understand this term. It follows that no one who is engaged in any special branch of medicine or surgery can afford to neglect a survey of the body as a whole, mental as well as physical, if he is to do the best for his patient. But the specialist has a further duty to perform beyond the immediate need of his patient, and that is to bring before his fellows, who are engaged in the engrossing and exacting work of general practice, various facts and theories which may aid him in the difficulties he may be confronted with from time to time in the daily routine of his work. Of all the organs of the body the eye is one whose functions are more readily impaired than any other by disease in other organs or regions. The eye may afford the first symptom of disease in some other organ, near or remote. Amongst these diseases

some of the most important are those which affect the nasal accessory sinuses.

Before entering into a description of the symptoms of disease in these sinuses, so far as they affect the eye and orbit, it is necessary to have clearly before us the anatomical relations which exist between these sinuses and the orbit. The walls of the bony orbit in which the eyeball and its appendages lie is made up of many bones. On the inner wall are to be found from before back: (1) The nasal process of the superior maxilla. This bone may very occasionally help to enclose the most anterior ethmoidal cell. (2) The lacrymal bone. This is very thin and quite commonly covers in the anterior ethmoidal cells, and furthermore, by its union with the superior maxilla, contributes to the formation of the upper part of the lacrymal canal, the lower end of which opens beneath the anterior extremity of the inferior turbinate bone in the nasal cavity. (3) The ethmoid (orbital plate). This encloses the anterior, middle, and posterior ethmoidal cells. The upper end of the orbital plate is united to the orbital plate of the frontal, which forms the roof of the ethmoidal cells. The orbital plate of the ethmoid varies in thickness and may be exceedingly thin. In the line of suture, between it and the frontal, are to be found two canals—the anterior and posterior ethmoidal canals. The former transmits the anterior ethmoidal artery and nasal nerve, and forms a communication between the orbit and ethmoidal air cells and cranial cavity. The latter transmits the posterior ethmoidal artery and a few fine nerve twigs. It connects with the ethmoidal air-cells alone. Behind the ethmoid, at the apex of the orbit, is (4) the orbital plate of the palate bone; this commonly encloses a posterior ethmoidal cell. We see, then, that this inner wall is of great importance, for the extensive ethmoidal cell spaces are only separated from the orbit by very thin bony walls, and the cells themselves are in direct communication with the nasal cavity by orifices, which open into the superior and middle meati. The roof of the orbit is formed by the orbital plate of the frontal bone. In the anterior part of this bone lies the frontal air sinus, a space which occupies

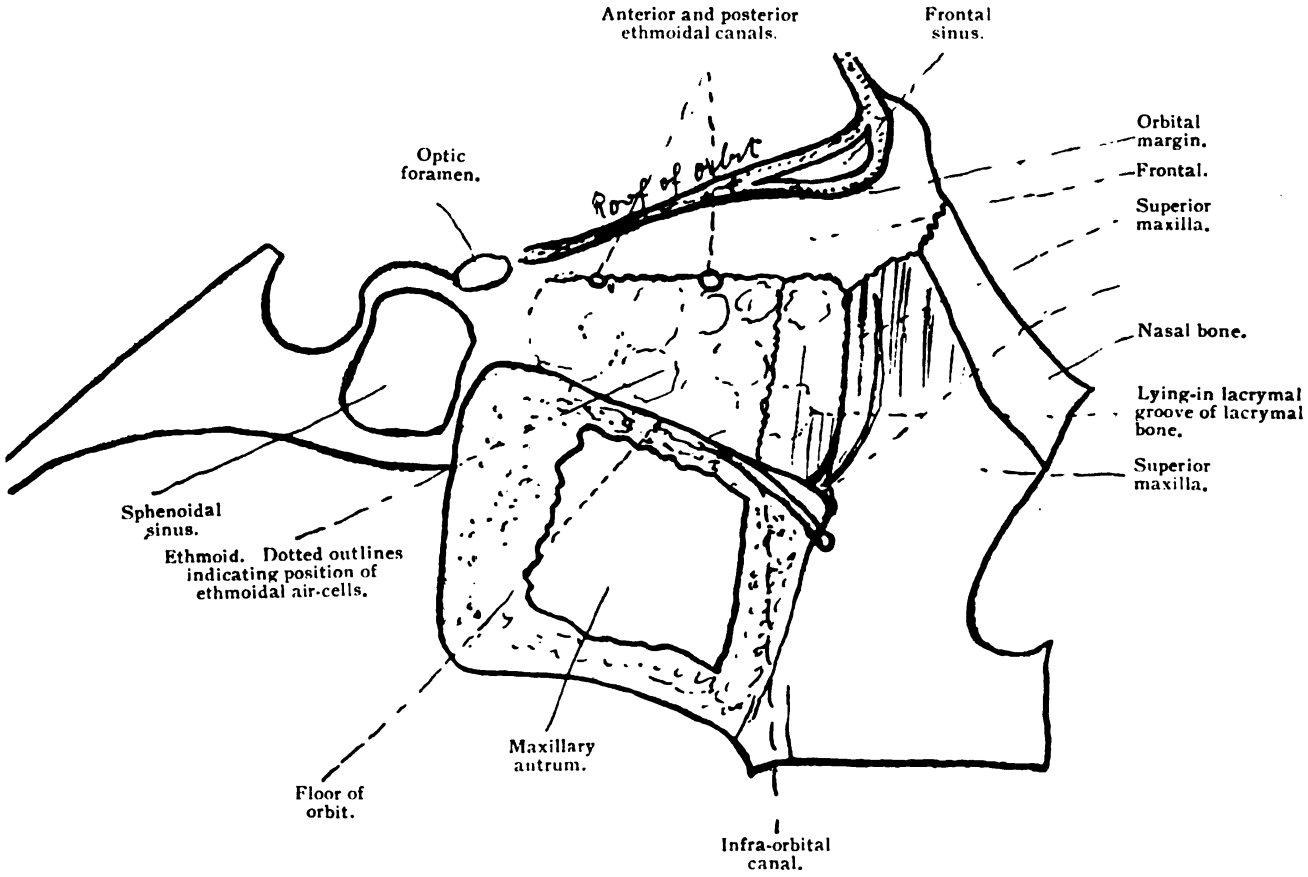

FIG. 1.—Oblique vertical section passing from apex to centre of anterior orbital margin (diagrammatic).

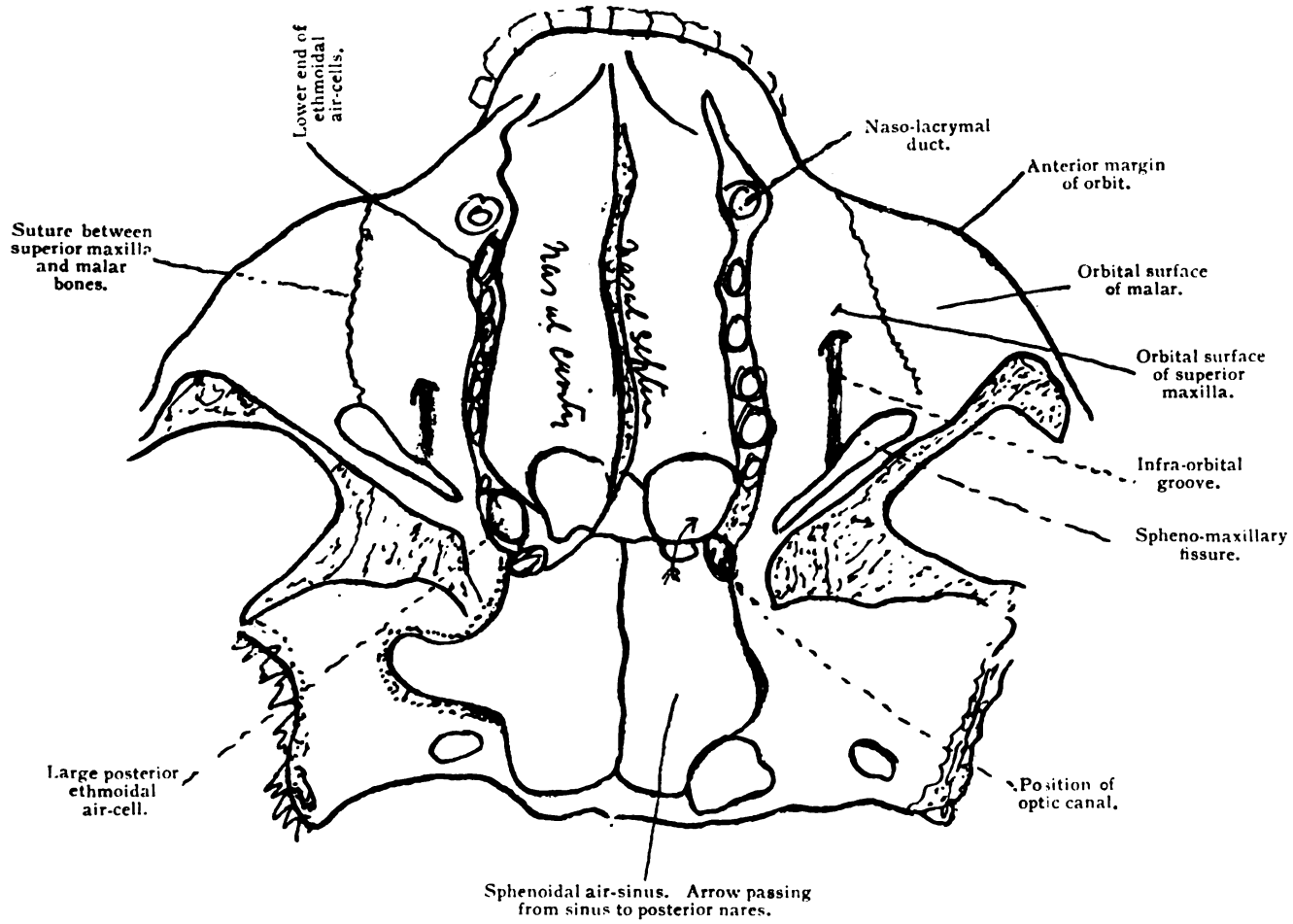
Lower end of ethmoidal air-cells.
Naso-lacrymal duct.
Anterior margin of orbit.
Suture between superior maxilla and malar bones.
Orbital surface of malar.
Orbital surface of superior maxilla.
Infra-orbital groove.
Spheno-maxillary fissure.
Large posterior ethmoidal air-cell.
Position of optic canal.
Sphenoidal air-sinus. Arrow passing from sinus to posterior nares.

FIG. 2.—Horizontal section through orbit (lower part).

the interval between the two tables of bone, and lies at the junction between the frontal and orbital portions. The frontal sinus varies much in size, gradually increasing with age. It may extend some distance back into the roof of the orbit, and the sinuses on each side are separated by a bony partition. The partition is seldom mesial, generally inclining more to one side than another. Occasionally it is even perforated, in which case one sinus will communicate directly with its fellow. The space opens at the inner extremity by a bony passage—the infundibulum—into the anterior part of the middle meatus of the nose. The anterior ethmoidal cells themselves also open into the lower end of the infundibulum. The floor of the orbit is mainly formed of the orbital surface of the superior maxilla, being completed on the outer and anterior part by the orbital process of the malar bone. Beneath the floor lies the large air space, the maxillary antrum. The bone which separates this cavity from the orbit is of variable thickness, and at times may be exceedingly thin. Traversing the floor of the orbit from behind forward is the infra-orbital groove, converted into a canal at the anterior end of its course. In this lies the infra-orbital nerve and artery. Finally, at the apex of the orbit, is the anterior extremity of the optic foramen, which lies on the upper and lateral aspect of the body of the sphenoid bone. It is to be remembered that the body of the sphenoid is a mere shell of bone enclosing a cavity—the sphenoidal sinus—which, like that in the frontal bone, is divided into two halves by a more or less median partition, which also may be perforated. The plate of bone intervening between this cavity and the optic nerve is never very thick and may be as thin as tissue paper. These, then, are the important points to be borne in mind in connection with our subject

To recapitulate: on the inner side of the orbit there lie the ethmoidal cells, separated only by a very thin lamina of bone. They may extend upwards and outwards into the roof of the orbit and downwards into the floor of the orbit and are of very varying size. These cells have a direct communication with the orbit by means of the anterior and posterior ethmoidal canals, and also by the lines of suture between the bones which form the inner wall of the orbit. The posterior ethmoidal air cell may be in close contact with the optic foramen. The roof of the orbit contains the frontal air sinus, which, however, has no direct communication with the orbit. The floor separates the orbit from the maxillary antrum, and it is noteworthy that in the floor there courses the infra-orbital nerve and artery from which twigs descend in the anterior wall of the antrum to supply the bicuspid and incisor teeth. Finally, at the apex of the space lies the sphenoidal sinus, having on its outer wall the optic foramen, in which lies the optic nerve. We see, therefore, that the orbital cavity is surrounded by these air spaces, with the exception of its outer wall.

We will now pass on to consider some of the clinical sequelæ of diseases of these sinuses. I shall deal with the subject mainly from the point of view of the eye and its adnexa, and shall touch but very lightly upon the pathological changes in the sinuses themselves, or the ætiology of the diseases with which they may be attacked. Broadly speaking, such diseases group themselves into two classes: (1) Acute or chronic inflammatory conditions, (2) new growths. Each of these groups may cause definite changes within the orbital cavity, which may be of an inflammatory type or mechanical.

The conditions arising in the eye and orbital cavity as the result of sinus disease may be grouped for convenience of description under the following headings:

(1) Affections of sight. This may be (*a*) manifest or (*b*) latent.

(2) Defects of movements of the eye.

(3) Changes in position of the eye.

(4) Inflammatory conditions of the orbit.

(5) Affections of the tear passages.

(6) Inflammatory affections of the eye itself.

I am aware that this is, perhaps, not a very scientific classification, but it will at all events serve to bring out the main features that result from sinusitis so far as they affect the eye and orbit. Furthermore, it must be carefully borne in mind that symptoms in disease can seldom be grouped satisfactorily, overlapping must occur, for two or more symptoms may occur together, and one may be dependent on another.

(1) In this first group I have included two subdivisions: (*a*) *manifest disturbance of vision.* In some cases this may be nothing more than a feeling of tiredness in the eyes when reading or other close work is being done. It comes on soon after any work has been taken up, and may be quite independent of any errors of refraction. It is, in fact, a true asthenopia. These cases are naturally not at all easy to recognise at first sight, but where the symptoms persist after any refractive error or other ocular abnormality that may be present has been corrected, and where there is no other obvious general condition such as anæmia, neurasthenia, etc., to account for the trouble, careful examination of the nose and its accessory sinuses should be made. The explanation of these cases is not obvious, but it is worthy of note that the eyeball and the nose have the same sensory nerve supply.

The nasal nerve, after passing through the orbit and supplying the two long ciliary nerves to the eyeball and the sensory root to the lenticular ganglion, passes through the anterior ethmoidal foramen, and after a short passage in the cranial cavity gains entrance to the nose. It is possible, therefore, that the asthenopia may be of a reflex nature. More important than these asthenopic symptoms is the interference with the visual acuity.

The failure of visual acuity is, to begin with, at any rate, unilateral, and it may be slight or very pronounced, so that the patient is totally unable to read with the affected eye. If slight the patient often complains that as soon as reading is started the print becomes confused. This loss of sight may be sudden or gradual; often it is not noticed until more or less suddenly reading becomes difficult or impossible, indicating perhaps involvement of the opposite eye. This is generally associated with symptoms belonging to the next subdivision, which I have labelled latent, and which I will now discuss.

(*b*) *Latent visual defects.*—Under this term I include disturbances of the visual fields either in the form of a general concentric contraction or of

central scotoma for colours. The latter is the more frequent condition, and it is the one which accompanies and often causes the defect in sight. This is so frequent that its presence in a *woman* is strong presumptive evidence of the existence of disease of the posterior nasal accessory sinuses, *i. e.* the posterior ethmoidal or sphenoidal cells. I have advisedly used the word woman, because in men there is yet another disease which may produce a well-marked scotoma to red and green—I refer to tobacco amblyopia, a condition which is exceedingly prevalent in this district where there is a large mining population in the surrounding areas, together with a large sea-faring element in the city. It is probable that a central scotoma is a very frequent symptom in diseases of the posterior nasal accessory sinuses, and is therefore of considerable importance. Its presence is very easily elicited in the following manner: Let the patient stand or sit before you with his back to a good light. Cover one eye with a shade or anything convenient, and with the uncovered eye let him fix that of yours which is opposite to his uncovered eye. Take a small red disc about 5 mm. in diameter and fix it at the end of a pin or point of a knife, etc. Holding the disc about midway between your own and the patient's eye, move it round about the fixation point in gradually widening circles until the blind spot is reached. If a scotoma is present the colour will disappear over a small area to reappear again when the disc is moved away. This scotoma generally lies close to the fixation point and slightly to the outer side, though it may appear at other points and sometimes takes the form of a ring round the point of fixation. Of course, care must be taken that the patient keeps his eye fixed steadily upon your own during the examination.

The scotoma may be absolute or relative, *i. e.* when absolute the colour disappears entirely, but when relative the colour does not disappear entirely, merely becoming fainter. In relation to the existence of the colour scotoma is the presence of an enlargement of the blind spot, an early and important sign, but one which requires careful examination with a perimeter or scotometer for its elucidation, and is therefore somewhat outside the scope of the general practitioner. If the enlargement of the blind spot is great it is, however, possible to detect it in the same way as the scotoma, comparison being made with the blind spot of the examiner. It not infrequently happens when the scotoma is large for it to run out and become merged in the blind spot. In advanced cases the scotoma may be present for white as well as colours. As indicated above, other conditions may give rise to central colour scotoma, besides diseases of the accessory sinuses. It is not necessary, however, to go into the question of the various causes of scotomata here, and I will merely mention two other causes which may be met with: (1) tobacco amblyopia, as already mentioned, and (2) the early stages of disseminated sclerosis. In these two diseases the affection is commonly bi-lateral, whereas in sinus disease the scotoma is usually unilateral, though this is by no means invariable.

The *visual fields*. The visual field may be much restricted, but there is nothing characteristic in its form. When the close relation which exists between the sphenoidal sinus and the posterior ethmoidal cells and the optic foramen is remembered, it is not surprising that the optic nerve may show signs of disturbance in its functions; for a concentric contraction of the visual field is evidence of disorder or disease of optic nerve. In the cases we are considering this disturbance may be due to a toxæmia or from direct involvement of the nerve itself by inflammatory changes spreading from the diseased mucous membrane. The more highly organised fibres which run from the macular region (papillo-macular bundle) are those most likely to be injured first, hence the frequency of a central scotoma; later the whole nerve becomes involved and so contraction of the visual fields occurs. It is not only in diseases of the posterior nasal sinuses that these changes in the visual fields occur, for they may also occur in disease of other nasal sinuses. I have recently seen a case of a boy, æt. 15 years, who suddenly began to lose vision in the left eye. On examination it was found that there was considerable contraction of the visual field with a marked paracentral scotoma to red and green. Ophthalmoscopically there was found a slight optic neuritis. Examination of the nasal cavity revealed an empyema of the maxillary antrum. Following treatment of the antrum, his symptoms all gradually disappeared, the fields becoming normal, the scotoma being completely lost, and the optic neuritis subsided. In this case no other sinus was involved. We see from

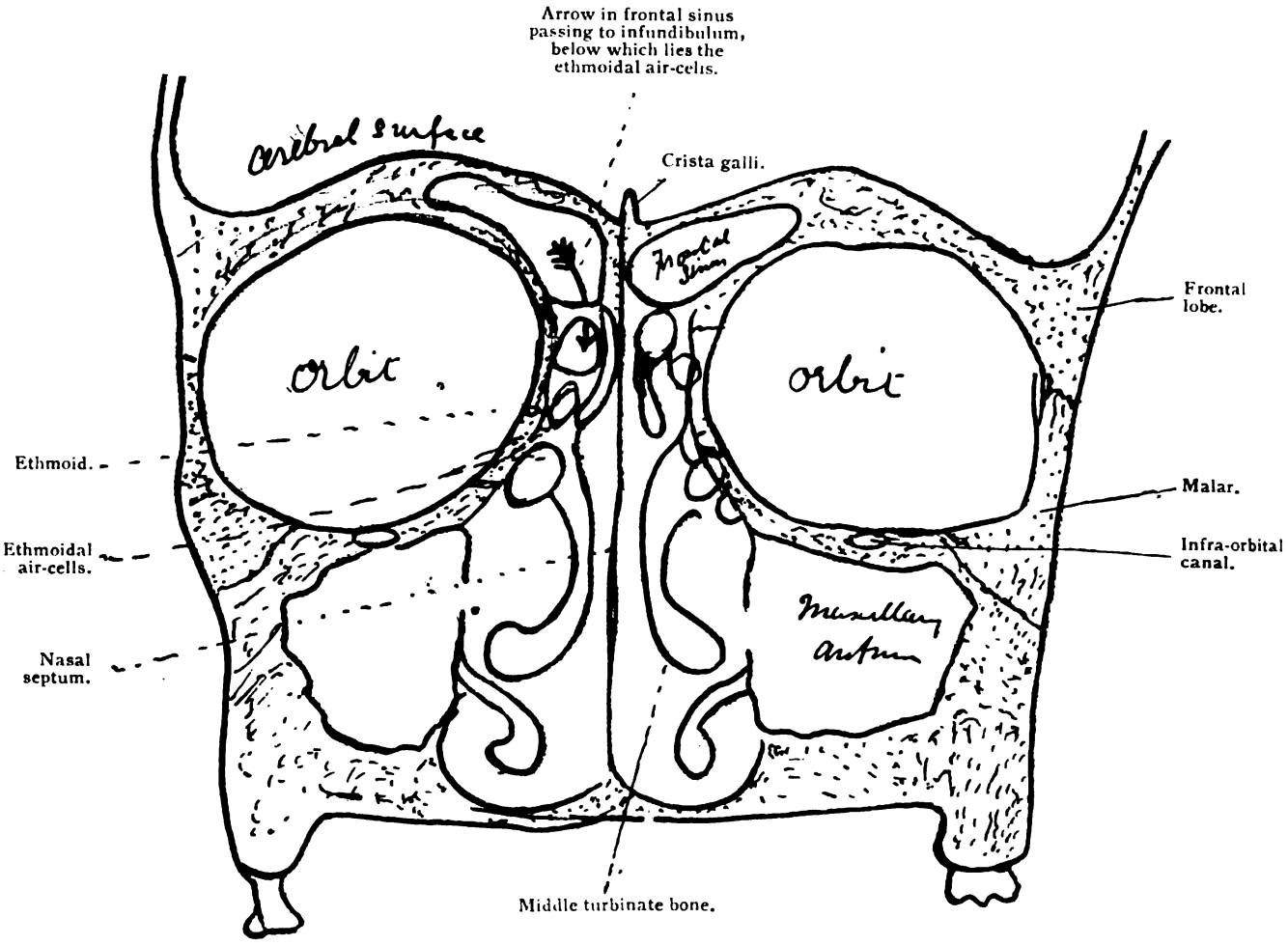

FIG. 3.—Vertical transverse section through orbits just behind orbital margin.

this case that, associated with affections of the sight in the visual fields, there may be definite optic neuritis. Often, however, ophthalmoscopic examination is negative. In my experience the evidence of optic neuritis seldom goes further than some increased redness of the disc with slight blurring of its edges, and I have never seen marked swelling of the disc, which, however, undoubtedly may be present.

(2) *Defects of movements of eyes* —These are by no means common. There may be a definite paralysis of one or more muscles, or, what is stated to be more common, a paresis of one or more muscles may exist, which upsets the muscular balance of the eye and so leads to vertigo and other symptoms depending on this condition. These latent weaknesses of a muscle come under the term heterophoria (latent squint). The muscles may be involved directly by extension of the inflammatory process from the sinuses to the orbit in one of the ways which will be discussed later, or the muscles may be affected indirectly through the nerves. In the latter case the fourth nerve is the one most likely to be involved, because in its course to the superior oblique it lies close to the posterior ethmoidal cells. From time to time one meets with cases of sudden paralysis of an ocular muscle in a person in whom there is no evidence of any general nerve lesion, *i. e.*, tabes, etc. Many of these cases clear up rapidly, but others are more enduring. It is quite possible that these are not "rheumatic" in origin as they have been labelled in the past, but are the result of some pathological condition in the nose or its accessory sinuses, transient or permanent. The term rheumatic is slowly becoming less frequently applied as an ætiological

factor as our knowledge of diseased processes increases; and when we use it probably few of us have any clear conception of what we mean. Defects of movement may also accompany other symptoms such as changes in position of the eye, which I will next discuss, and may be the result of simple mechanical disadvantage.

June 7th, 1915.

(*To be concluded.*)

Tetany following Biliary Colic.—G. Graul can find no other record of the association of tetany with biliary colic, which he observed in the following case.

A young married woman, fat and neurasthenic, had suffered for some weeks from abdominal pain, which was attributed to cholelithiasis. At the height of an attack of biliary colic she developed typical tetany, which lasted about half an hour. Both hands were fixed, as in writer's cramp, and, though the contraction of the fingers could be overcome, they returned to this position as soon as they were released. Pressure over large, detached arteries or nerves caused muscular spasm, and the facial nerves reacted with abnormal readiness to mechanical stimulation. The fingers, hands, and forearms were livid and paræsthetic. Movements were painful, and the patient was exceedingly restless, anxious and excited. The tetany passed off with the colic and did not recur, but the patient was jaundiced for three days after the attack, which was followed by the passage of three large gall-stones *per rectum*.

Probably the irritation caused by the passage of large stones through the bile-duct contributed to the reflex provocation of tetany. But as biliary colic is common, and is practically never accompanied by tetany, there must have been another factor in the case. This factor, the writer thinks, was a predisposition to tetany, due to faulty action of the parathyroids, for abnormalities of these glands have for some time been held responsible for certain nervous disorders, including tetany.—*Deut. Med. Woch.*, p. 249.

CLINICAL JOTTINGS.

(*Continued from p. 253, vol. xliii.*)

By SAMUEL WEST, M.D, F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

Forcible Movement of Stiff Joints.

The forcible moving of stiff joints can do harm as well as good. The breaking down of adhesions, once so popular, is rarely practised now, for the violence necessary causes so much pain and does so much injury that the movements cannot be kept up—the adhesions reform, and the limb simply becomes stiff and fixed in a new position. Instances of the immediate cure by the breaking of an adhesion—the stock-in-trade of the professional bone-setter—are very rare indeed.

Passive movements short of violence are often beneficial, yet they, too, may retard rather than accelerate progress. The stretching of stiff joints causes pain at the time, but the pain passes off so soon as the stretching is discontinued; if not the treatment is doing harm. It is wonderful what gentle movements patiently persisted in will do if they produce only the sense of stretching short of actual pain. Pain which lasts after the movements are stopped is the indication that no good will come of the treatment, and the length of time the pain continues is the direct measure of the harm which is being done.

Forms of Arterial Thickening.

Arterial sclerosis is merely the Greek translation of thickened arteries, yet it is often used as if it were a definite pathological condition and not a general term.

The two common forms of arterial thickening are due to atheroma and granular kidney, and they should be kept distinct and not confounded.

Atheroma is a widespread degeneration occurring more or less in everyone as age comes on, affecting many vessels, especially the aorta, the coronaries, the arteries of the brain, and in general the large and medium-sized arteries. Though widespread, it is not universal. It is patchy and irregular.

The arterial changes in *granular kidney* are universal, affecting all the arteries of the body, large and small, and more or less uniform—that is, not patchy. They are met with frequently in middle

life when atheroma is uncommon, or at a still earlier age when atheroma is practically unknown.

Syphilitic disease of the arteries is essentially local; affects, as a rule, a few vessels at the most here and there; may occur, of course, at any period of life, but is chiefly met with in the young adult. Widespread syphilitic disease of the arteries is rare. The radial at the wrist is practically never affected, so that if that artery be found thickened, the thickening there is almost certainly not syphilitic, but due to some other cause.

There is another form of thickening in the young to which no reference is usually made, but it is important. It is met with *in athletes*, that is to say, in perfectly healthy persons of great muscular power. It is a hypertrophy of the vessels like that which occurs in such persons in the heart—a true muscular hypertrophy. After lecturing on thickened arteries, it has frequently happened to me that some fine specimen of muscular humanity among the students has come to me with great concern because he had found his own arteries thick. I have consoled him with the statement that if they did not hypertrophy he could not be the successful athlete he was. This hypertrophy of the vessels may come and go like that of the heart, according to the work demanded.

(*To be continued.*)

Treatment of Prolapse of the Rectum.—Roux advocates anew his method of treating prolapse, the advantages of its simple technique having been amply proved. He injects alcohol into the connective tissue around the rectum, outside the intestinal wall. Under general anæsthesia the needle is inserted back of the rectum, on the median line, and alcohol is deposited here and on each side, between the rectum and coccyx and sacrum. The needle is then introduced again on each side farther around, but the pouch of Douglas is respected. He has thus injected from 2 to 4 or 6 c.c. of alcohol in children, with no untoward results, not even when up to 10 c.c. was injected. A roll of gauze smeared with petrolatum is placed between the buttocks and brought up entirely around the abdomen, holding the rectum firmly in place. This is left for three to eight days or more. Adults can be kept constipated for this period, but defæcation has to be permitted to children.—*Journ. A.M.A.*, lxiii, p. 1242.

Myiasis of the Urinary Passages.—E. F. King reports the case of a man, æt. 32 years, who, on October 27th, brought an object that he had passed with the urine, which was later identified as a larva of *Fannia scalaris*. He had voided similar objects with the urine October 19th and 28th. He had chronic posterior urethritis and for some months had been in the habit of micturating into a glass, which he kept in the shed at the rear of his house, turned upside down on a shelf, and it was always rinsed after using. He had been using a small syringe of hard rubber for urethral injections, and had kept this wrapped in paper and in a closed drawer, so that, apparently, the larvæ could not have been in the glass, nor the eggs or larvæ in the syringe.

About 8 o'clock in the morning, while digging, he was seized with an intense desire to micturate, felt something pass with the urine, and found the larva in the glass. He has seen no larvæ since, nor was he able to find any in the fæces after diligent search.

Contamination probably took place while at stool, the flies being attracted by the slight urethral discharge. That larvæ were voided ten days apart would indicate that they were from different infections. They had evidently penetrated the prostatic urethra, judging by the patient's sensations, though the cystoscopic findings were negative.

Réné Chevrel in 1909 analysed all reported cases of myiasis of the urinary passages and added a case of his own. Only twenty such cases had been reported, six of which he regarded as authentic, ten as probable, and four as doubtful. He was of the opinion that while dipterous ova may be carried into the urinary passages by sounds or catheters, or in water used for irrigating, they generally are deposited about the meatus urinarius, where are found the conditions favouring their growth—darkness, moisture, warmth, oxygen, and food, which last is furnished by a gonorrhœal or other albuminoid discharge. The eggs hatch promptly, and the young larvæ pass into the urethra and on into the bladder in search of food. They require but little oxygen, and if kept under water for hours become active when taken out.—*Journ. A.M.A.*, lxii, p. 2285.

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We regret that owing to the war the number of pages must be diminished for a time.

THE STATUS LYMPHATICUS.*

By HUGH THURSFIELD, M.D., F.R.C.P.,
Assistant Physician St. Bartholomew's Hospital; Physician for Out-patients, Hospital for Sick Children, Gt. Ormond Street.

IN 1905 the Society for the Study of Disease in Children devoted a sitting to the discussion of the causes of sudden death in children, and among other phenomena gave consideration to the subject of our discussion this afternoon. In the following years the literature upon status lymphaticus has grown to alarming proportions, and every year adds an increasing number of communications. But in spite of this fact I am afraid that our absolute knowledge of the condition remains almost as small as then, though I think that the limits of our knowledge are better defined, and that there is less tendency to stray into somewhat vague speculation.

I must first recall the facts which led up to the formulation of the conception of a constitutional disorder called "status lymphaticus." Away back in the seventeenth and eighteenth centuries our predecessors recognised that there occurred deaths which could be attributed to the pressure of a large thymus gland upon the vital structures contained in the upper aperture of the thorax. It is probable that the earliest recorded cases were examples of malignant disease of the gland, but in the nineteenth century it was recognised that there was a group of cases of dyspnœa terminating with sudden and unexpected death in which the only anatomical morbid lesion was the presence of an abnormally large thymus. In 1850 Friedleben, after a detailed research into the anatomy of the gland, came to the conclusion that there was no such thing as thymic asthma. That this dogma, like most dogmatic statements on controverted

Read at the Royal Society of Medicine.

questions, was exaggerated is, I think, now quite clear; but it cleared the ground for the conception formulated by Paltauf in 1889 of a constitutional disorder manifested by a hypertrophy of all the lymphatic structures of the body, including the thymus gland—a condition to which he gave the name of "status lymphaticus." The features upon which he laid stress were the enlargement of the thymus gland, the hypertrophy of the tonsils and the lymphatic tissues of the naso-pharynx, the enlargement of the lymphoid tissue at the root of the tongue, the hypertrophy of Peyer's patches and solitary follicles of the intestine, the large spleen. To these he added the hypoplasia of the aorta, a feature which has seldom been observed since. Such abnormalities he held to be the outward expression of a constitutional disease which rendered its possessors peculiarly liable to infection and to sudden failure of the vital functions.

Paltauf's hypothesis has had vigorous critics and valorous supporters; both, if I may venture my own opinion, undiscriminating and indiscreet. But we are to-day in a better position than in the discussion of 1905 as regards the knowledge of the normal anatomy of the thymus gland. Then it was commonly stated that the gland reached its maximum size in the last months of fœtal life and during the first two years of infancy; and that after this date it rapidly atrophied. Even now, however, it remains a disputed point what is a normal weight for the gland at any given period. In a paper which I wrote in 1902 I gave reasons for believing that the normal weight during the period of infancy had been considerably exaggerated, and Dudgeon in 1995 came independently to the same conclusion. We considered that any thymus gland which exceeded 10 to 12 grm. during the first two years of life must be considered abnormal.

I must, however, add that some of the German authorities put the normal weight much higher, at 25 grm., and that one of the more recent text-books of anatomy in this country states that the gland at puberty may be merely remains, or may weigh as much as 40 grm. Morbid anatomical experience, however, at least in this country, does not agree with these statements, and it is exceptional to find a thymus gland of more than 10 grm. except in connection with the conditions I am about to mention.

Friedleben in 1850 maintained: "Es gibt kein Asthma thymicum." It now, however, seems to be generally agreed that he made a mistake, and that there are cases of dyspnœa which are directly attributable to the pressure of the enlarged thymus. For example, Klose, writing in 1911, was able to bring forward nineteen cases in which the excision of the whole or a part of the thymus had relieved the dyspnœa; in another case the dyspnœa persisted, and in three others the operation was fatal. He came to the conclusion that the cause of such dyspnœa was pressure by the enlarged gland and that all such cases "belong to the surgeon." Parker, an American author, in 1913, collected fifty cases in which operation had been performed for the relief of dyspnœa. The mortality of the operation was considerable (seventeen deaths, 33·3 per cent.), but the deaths were seldom to be attributed to the operation. It should, however, be noted that in thirteen of these seventeen cases there was infection either of the wound or of the lungs. Even if we take only the cases recorded since the beginning of 1910, nine out of twenty-three patients were dead within a month of the operation, while of the recoveries one required a second operation six months after the first; the relief from the dyspnœa was generally gradual and not immediate, though in about half the cases the acute attacks of suffocation disappeared at once. My own experience is that these cases of so-called thymic dyspnœa are extremely rare. I have been on the look-out for such cases for many years, and I have asked many friends of greater experience than mine, but with one exception I have yet to meet with symptoms at all suggesting the condition. This case was that of a baby, æt. 5 months, who had a history of making gurgling noises since birth and of becoming very blue when he cried. He had some inspiratory and expiratory stridor and some dilatation of the veins of the neck and cyanosis when he cried, but no recession of the chest walls and no bulging tumour in the episternal notch. On examination he had an area of impaired resonance over the manubrium sterni, and for a distance of about $\frac{1}{2}$ in. on either side. The dulness was continuous with the heart dulness. An X-ray picture showed an opaque shadow coincident with the dull area, such as might be caused by a bulky thymus gland. Intubation of the larynx did not relieve the stridor. Thymectomy was discussed, but before a decision was made a reddened area appeared above

the inner end of the right clavicle, and an abscess was opened, in connection presumably with the deep lymphatic glands, with complete relief of the dyspnœa. The cavity of the abscess extended backwards to the vertebræ, and the trachea was displaced to the left side.

My own opinion is that in many of these cases of dyspnœa the thymus is not responsible for the pressure, but that, as in this case, the real source is the enlargement of deep glands. And I must add that in the records of the fatal cases following operation there is no statement of the condition of the lymphatic apparatus elsewhere in the body; that is, there is no attempt to establish the diagnosis of lymphatism, while in several of these the tracheo-bronchial glands are described as necrotic.

But, whatever may be our opinion about these dyspnœic cases, there can be no doubt that pressure plays no part in the cases which I am next going to consider, the sudden deaths, whether in infants who have given no previous signs of disease, or in those who are suffering from some trivial disorder, or again, in patients who expire without warning under an anæsthetic. It is a common experience of the post-mortem room to make an examination of the body of a baby who has been found dead without any previous illness, and in the bulk of these the morbid anatomy is constant—an enlarged thymus, usually between 25 and 35 grm., bulky and thick, often covered with small petechial hæmorrhages, a hypertrophy of the Peyer's patches and of the solitary follicles of the intestine, and an enlargement of the mesenteric, bronchial, and cervical glands. It has been maintained that such cases also are due to the pressure of the thymus upon the vital structures. The hypothesis takes various shapes; sometimes that the pressure is exercised on the trachea, and that this becomes flattened; but it has been shown that an elliptical shape is not uncommon in the normal child. Then, that the pressure is exercised on the great vessels; but there is no sign of engorgement of the organs such as would accompany such pressure, and often the heart is empty and firmly contracted. Lastly, that the pressure acts upon the vagi and inhibits the heart's action. I was some years ago an eye-witness of such a death. A boy, æt. 13 months, "suddenly sat up, his eyes became fixed, he ceased to breathe, became very slightly blue, and then quite white, and fell back dead. The whole series of events occupied less than thirty seconds." Such a manner of death does not suggest pressure. Bellamy Gardner's experience with a boy, æt. 13 years, who was under chloroform, was very similar. The patient, just after the operation had been begun, "made a slight retching movement . . . stopped breathing . . . a deep navy-blue coloration appeared on the forehead and temples . . . the lips and ears were at the same time almost normal in colour . . . From the moment of respiratory paralysis not one single natural muscular or respiratory movement took place." In both these cases the autopsy showed the presence of the classical signs of status lymphaticus, and the complete absence of any signs of obstruction to the respiratory passages, and, further, the absence of any signs of pressure upon the great veins of the neck. If pressure be the cause of death, it appears to me that it is inconceivable that the great veins and sinuses of the brain should show no signs of dilatation and congestion. Before leaving these cases I must add that such "anæsthetic" deaths are, in a very large number of instances, due to the existence of this mysterious condition. Spilsbury, who has had an unusually wide experience, has stated that the majority of sudden deaths during anæsthesia which he has had to investigate were associated with the presence of status lymphaticus. Nor do they occur with chloroform alone. Of thirty such cases McCardie found that in seventeen the anæsthetic was chloroform, in six ether alone was used, and in five others a mixture of ether and chloroform. Further, in two cases local anæsthesia had been employed, in one cocaine, in the other Schleich's infiltration method. Nor is the death due to the shock of the operation, for in more than one instance the operation had not been begun. Lastly, in several the patients had been safely anæsthetised, the operation had been completed, and the patient back in bed had recovered consciousness, and then when all seemed safe suddenly death had supervened. Such deaths are quite unlike the so-called "delayed chloroform poisoning," in which condition there is seldom any suddenness in the fatal issue, but profound collapse with vomiting and acetonæmia fill a more or less prolonged interval.

Besides these two well-recognised groups some writers have found evidence of status lymphaticus

in sudden death occurring in the course of certain diseases which do not usually lead to a fatal issue. The most striking of these is the sudden death which occasionally occurs in the eczematous conditions of infants. Feer in 1904 was able to collect thirteen cases of death during eczema, and in ten of these there was an enlarged thymus and hypertrophy of the lymphatic structures. He does not contend that all deaths in eczema are associated with this condition, since some are undoubtedly due to an acute infection. It is interesting in this connection to recall that Gardner's patient had suffered from obstinate eczema before his death.

Some authors have also maintained that a status lymphaticus is often found in men who have committed suicide, but this is certainly not a common experience.

Of other diseases in which an enlarged thymus occurs I will merely mention myasthenia gravis, a disease which sometimes ends in attacks of dyspnœa and respiratory failure, Addison's disease, and leukæmia. In these two last the enlargement of the gland is by no means common. A more important association is that with Graves's disease. Here it is rare for an autopsy to be performed without finding a considerable enlargement of the thymus. Capelle found that of twenty-two cases of exophthalmic goitre dying under an anæsthetic twenty-one had an enlarged thymus gland. Although the other signs of the lymphatic state are often not marked, it is probably to the coexistence of this condition that we must attribute the well-known danger of general anæsthesia in Graves's disease. At the same time I do not think that apart from anæsthesia sudden death is common in this affection.

The Origin of the Lymphatic State.

When we pass from the consideration of the manifestations of lymphatism to that of the origin of the condition, we pass into a region of vague speculation where very little is known, and that little very inaccurately. In recent years Basch, and later Klose, have conducted a series of thymectomies on puppies, and though it can hardly be said that these experiments throw much light on the lymphatic state, they do at least widen our conception of the importance of the thymus gland and of its wide connections with the metabolism of the body, especially in the period up to puberty. Basch and Klose have found that when the thymus gland is extirpated in puppies quite early in life the animals grew more slowly than the control animals. Their muscles were more flabby, their bones were softer and more liable to bend and break, their intelligence was less, and after the fourth month they rapidly passed into state of coma and died. Microscopic examination of the bones showed the characteristic changes of rickets. Klose carried out elaborate examinations of the calcium excretion in these animals and showed that the tissues of the experimental animal were markedly deficient in lime salts. He believes that the thymus is chiefly engaged in hindering the formation of, and in neutralising the excess of acids in the organism, and that its enucleation leads to an acid excess in the system. He states that some of the symptoms in the animals remind the observer forcibly of the manifestations of tetany in young children. These observers and others have found that there is an intimate connection between the thymus and the testes, which tend to enlarge by the growth of fibrous tissue in them after thymectomy. Miss Hewer has lately obtained somewhat similar results in a series of rats fed upon fresh gland substance; the male rats showed retardation of sexual maturity and often degeneration of the testis. Other experimental work appears to indicate that there is a more or less intimate connection between the thymus and the other glands of internal secretion: the suprarenals, the thyroid, and possibly the pituitary. These deductions, however, rest at present on insufficient grounds.

The net result of these investigations is, I think, to widen our conception of the functions of the thymus, and incidentally of status lymphaticus. This condition is not merely a disorder of the thymus, but a much more complicated disorder of the whole "ductless gland" system, and until we obtain a clearer conception of the inter-relations of these glands we shall not progress in our understanding of status lymphaticus.

Diagnosis and Treatment.

Meanwhile it is necessary to recognise the extreme importance of diagnosing the condition, since it so often leads to tragedy. The enlargement of the thymus is the most constant feature, and we must

endeavour to discover its existence in all cases of children who are to undergo an operation, especially in those who are the subjects of lymphoid hyperplasia. The most valuable means at our disposal is certainly the discovery of an increased area of dulness over the manubrium sterni. The normal impairment is said to be a V-shaped area scarcely transgressing the margins of the bone, while in enlargement of the gland this area passes for ½ to ¾ in. to either side, and below joins the cardiac dulness. Next to this the X-rays are of some value, but I do not think will distinguish between a thymus and enlarged lymphatic glands in the anterior mediastinum. If with these physical signs there is a tendency to unexplained attacks of dyspnœa or to a persistently low vitality, as evidenced by subnormal temperatures and intolerance of exertion, I think that status lymphaticus should be diagnosed and the child should not be exposed to the danger of an anæsthetic.

Dr. Leonard Williams, in the discussion on Gardner's paper, held that the status lymphaticus was, in reality, an expression of disordered metabolism on the part of the thyroid gland, and that the administration of thyroid extract would avoid many of the accidents met with in the course of anæsthetising children. While I cannot personally feel that there are any good grounds for the indictment of the thyroid in this condition, I yet recognise that it is almost certainly a disorder of the "ductless gland" system, and therefore that it is legitimate to expect something from the employment of an extract which we know has a profound effect upon this system. I should myself be more inclined to to use pituitrin, an extract which has the most extraordinary effect in obviating the results of shock; and to "shock" in a broad sense it appears to me that we may attribute the fatal results of the lymphatic state. Wherever I suspected the existence of the condition I should feel inclined to give an injection of this drug before administering an anæsthetic, and I should be glad to hear of the results of such administration on some of the children who are said to be the subjects of thymic asthma.

June 14th, 1915.

AFFECTIONS OF THE EYE AND ORBIT IN DISEASES OF THE NASAL ACCESSORY SINUSES.

By D. LEIGHTON DAVIES, M.S.Lond., F.R.C.S.,
Assistant Lecturer on Anatomy, University College, South Wales; Assistant Ophthalmic Surgeon, Cardiff Infirmary.

(*Concluded from p.* 191.)

(3) *Changes in position of eye.*—This group of symptoms is very commonly associated with the group that follows, but it may occur as an isolated symptom. The eye-ball may be displaced in various directions, directly forward or to one side, upwards, downwards, inwards, or outwards, or these may be combined in many ways. The direction of the displacement may give a rough clue to the sinus at fault. In diseases of the frontal sinus the direction is usually down and out; in ethmoidal disease when the anterior cells are alone involved outwards, but more usually it is outwards and forwards when the posterior and middle group of cells are involved. In sphenoidal sinus disease the displacement is usually forward or forward and outward. Associated with the displacement, as already indicated, there may or may not be some limitation of movement of the eye, and there may be diplopia. The forward displacement of the eye may be so great as almost to merit the term dislocation, but this, of course, is rare. When present the eye is practically immobile. Displacements of the globe may occur in the following ways:

(*a*) By extension of the inflammatory process to the orbit. This may be merely a periostitis, or it may be a cellulitis of the orbital tissues with or without abscess formation. Proptosis of this type is generally accompanied with some ptosis and œdema of the lids. On ophthalmoscopic examination some engorgement of the retinal veins will probably be found and perhaps slight optic neuritis.

(*b*) By reduction of the size of the orbital cavity. This may be brought about by the distension of one or other of the sinuses by fluid or new growth. Under pressure the thin walls bounding the orbital cavity yield or gradually atrophy till ultimately the swelling projects into the orbital cavity, and if due to a fluid distension of the cells is called a mucocele.

These are particularly common in the ethmoidal air cells.

(*c*) The orbital cavity may be invaded by some new growth, generally, of course, malignant, which arises in the sinuses. In these cases the displacement may be of the most extreme kind, so that the lids are incapable of affording a complete covering to the cornea. As a result, the cornea sooner or later ulcerates; this may be followed by panophthalmitis, ending in total loss of the eye. Loss of sight may also occur in these cases from involvement of the optic nerve by the new growth, though it is remarkable how the nerve may escape when completely surrounded by growth. Primary optic atrophy is the more common ophthalmoscopic finding.

(*d*) Another cause somewhat similar to that mentioned in group (*a*) is thrombosis of cavernous sinus. This is most likely to occur when the posterior ethmoidal or sphenoidal sinuses are the seat of septic trouble. As already pointed out, the cranial cavity is separated from the sphenoidal air cell by bone, which may be very thin, or, indeed, sometimes perforate, the result of caries or defective ossification. In either case infection can readily pass to the dura, which becomes involved in the inflammatory processes. The large venous space known as the cavernous sinus, the walls of which are formed by the dura mater, lies on the side of the body of the sphenoid, one on each side. The two sinuses are connected across the upper surface of the sphenoid by small channels known as the circular sinus, which lies in the pituitary fossa.

The ophthalmic veins drain into the cavernous sinus. Thrombosis of this sinus, therefore, leads to engorgement of the venous system within the orbit, and the protusion of the eyeball which results from this is always associated with distension of the veins of the conjunctiva, which assumes a dusky blue appearance. There is also usually distension of the retinal veins and possibly congestion and swelling of the disc with hæmorrhage into the retina. This form of proptosis is, of course, of very fatal significance, and usually ends in rapid death. The fundus changes described above may in part be due to the septic meningitis which nearly always accompanies thrombosis of the cavernous sinus when the result of septic sinusitis.

(4) *Inflammatory conditions of the orbit.*—For practical purposes it may be said that orbital inflammation is always secondary to disease of the accessory nasal sinuses. It has already been pointed out that displacement of the eyeball may be one of the signs of orbital inflammation, but it is not necessarily always present, and there are other signs. Perhaps the earliest is swelling of the eyelids; slight at first, and often appearing and disappearing rapidly, and thus simulating angio-neurotic œdema. Later, the swelling becomes greater, and oftentimes the skin has a dusky red appearance, and may have a brawny feeling. Owing to the swelling, the lids do not close efficiently, and the palpebral fissure becomes widened. There is also usually pain on movement of the eye and tenderness of the orbit to pressure. As the symptoms become more severe chemosis of the conjunctiva sets in. Orbital cellulitis is more likely to occur in diseases of the frontal or ethmoidal air cells, because the walls of the orbit in relation to these spaces are thinner than elsewhere. If an abscess develops it may be possible to detect it at the inner side of the eye as a fluctuating swelling. Frequently the formation of a fistula may be the first indication of an abscess. Where an empyema of the frontal sinus makes its way into the orbit the fistula is generally found in the upper lid rather to the inner side.

In a case recently seen, a fistula in the upper lid led back to a perforation in the anterior part of the roof of the orbit. A large quantity of pus was constantly discharging from the fistula. At the operation a large area of the floor of the frontal sinus was found to be necrosed and the sinus full of the most offensive pus and necrotic mucous membrane. The history dated back to an attack of influenza.

The inflammatory conditions which originate in the surrounding air spaces reach the orbit in one of two ways:

(*a*) The sinusitis causes suppuration of the mucous membrane, this in turn leads to osteitis and caries of the bony walls of the sinus, and so infection spreads to the periosteum of the orbit.

(*b*) Along the veins and their lymphatics: a septic thrombo-phlebitis of the veins being the means of extension of infection. There is no need for actual perforation of the walls of the sinus, as the lines of suture between the various bones afford passage to communicating veins of minute size as well as the larger vessels in the anterior and posterior ethmoidal canals

(5) We now come to a group of symptoms which depend on interference of functions of the tear passages, a group which is a well-recognised, frequent, and early symptom of sinus disease. In the first place, lacrymation may occur without obstruction of the drainage apparatus, and it is probably a reflex effect. This will be well understood when it is remembered that both the lacrymal gland and the greater part of the conjunctiva are ennervated by the ophthalmic division of the fifth cranial nerve as already described. A fistula in connection with the anterior ethmoidal cells or the frontal sinus may occur close to the lacrymal sac and so may simulate a fistula of the sac following lacrymal abscess. There is an important point of distinction between these two forms—in the case of a fistula in connection with the accessory sinuses its position is practically always above the level of the tendo oculi, whereas in the case of one of the sac itself it always lies below the tendo oculi without exception. A mucocele of the anterior ethmoidal cells may readily be mistaken for blenorrhœa of the sac, and, in fact, both may exist together. This is possible owing to the fact that the anterior ethmoidal cells are completed by the lacrymal bone, and they are frequently found in relationship to the upper part of the naso-lacrymal duct. From this it will be understood that enlargement of the anterior ethmoidal cells may cause mechanical obstruction of the duct and so leads to epiphora and later on blenorrhœa of the sac. Another cause of epiphora is the displacement of the lids, and consequently of the puncta, following displacement of the eyeball. A considerable part of the naso-lacrymal canal lies in the anterior wall of the maxillary antrum. It is, therefore, quite easy of comprehension that the naso-lacrymal duct may be affected by suppurating conditions in the antrum, and that this may lead to occlusion of the duct either by direct pressure upon it, by inflammatory effusion, or by cicatrisation of the duct following chronic inflammatory changes. This is, however, but theory, for the ordinary chronic obstruction of the lacrymal duct is not usually associated with obvious nasal or sinus disease. Such, at least, is the opinion of the majority of ophthalmic surgeons in this country, and with this opinion I am in agreement, for I have not yet met with a case of obstruction of the lacrymal duct which has been associated with or the result of nasal or sinus disease. There is, however, apparently a considerable body of opinion, more especially in America, which holds that, in the majority of instances, blenorrhœa of the sac or any obstruction of the naso-lacrymal duct is secondary to disease of the nasal cavity.

(6) The last group which includes inflammatory affections of the eye itself is perhaps less obviously associated with or dependent upon disease of the accessory sinuses. This may arise in part from the difficulty of assigning to a certain effect its own definite cause, which is particularly great in the deeper inflammatory affections of the eye. These cases also are always likely to be uncommon, for long before infection can have gone so far the presence of the offending sinus will probably have revealed itself by other more certain symptoms.

Practically all the tissues of the eye have been stated at one time or another to be involved as a sequence of nasal sinus disease. The cornea and sclera are, of course, liable to become affected, the cornea especially in orbital suppuration, and, as has already been pointed out, panophthalmitis may follow orbital suppuration. Iritis and irido-cyclitis are among the common intra-ocular diseases, but oftentimes the cause is very obscure. Undoubtedly some of these have their origin in a sinusitis, but they are not common. An example occurred in a male, æt. 42 years, who was under my care some time ago. He complained of irritation and pain of the left eye for four weeks, but this had got much worse during the ten days previous to his attendance at the hospital. He reported that the left eye had never been well since some polypi had been removed from the nose two years previously. There was marked iritis of the left eye. He was sent to the nose department, where it was found that he had suppuration of the ethmoidal cells on the left side. Treatment of these was followed by prompt relief of his iritis, and so far as I know he has remained well ever since. Diseases of the choroid are very rarely met with, and I have no personal experience of such cases, though it is not difficult to understand how an affection of the choroid could occur when the free communication which exists between the choroidal and ophthalmic veins is taken into consideration; indeed, it is somewhat surprising that association between choroiditis and sinusitis is not a common phenomena. The retina may be involved as part of an optic neuritis—neuro-

retinitis—with hæmorrhages into the retina, etc. Involvement of the optic nerve is the basis of the various disorders of vision which were enumerated under the first heading. Vascular disturbances of the retina may occur, thrombosis of the central vein or of one or more of its branches, and these cases, of course, are accompanied by more or less grave defects of vision according to the size of vessel involved. These, then, are the more common, or most marked symptoms which may arise in a case of nasal accessory sinus disease. It is a somewhat formidable list, but many of them are uncommon. It must not be forgotten that many of these symptoms may be produced by other than sinus disease, and it scarcely needs mention that not every case of sinus disease gives rise to ocular or orbital symptoms, for they are, comparatively speaking, uncommon. Yet to be forewarned is to forearmed, and it is to the interest of the practitioner no less than that of the patient that such cases should be promptly recognised and as promptly treated by those who are competent to do so.

June 7th, 1915.

Arteriotomy for Embolus in Brachial Artery.—Lundmark's patient was a woman, æt. 40 years, with chronic heart disease, but compensation had never failed. A short time after a felon, an embolus lodged in the brachial artery, where the deep brachial branches, and it was successfully removed ten hours after the first symptoms. In seven cases of arteriotomy for embolus on record, three proved successful, the interval ranging from three to seven hours. Key's patient was a man, æt. 43 years, and the embolus was at the fork of the femoral artery, and was successfully removed after seven hours. Bauer's patient was a man, æt. 39 years, and the large embolus at the fork of the aorta was successfully extracted after three hours. The only other successful case in his compilation was reported by Mosny, the embolus being removed from the femoral artery after six hours. In Lundmark's case the embolus extracted from the brachial artery was 3 cm. long and about 6 mm. thick. The embolus usually straddles a bifurcation, and the shorter the interval before the arteriotomy the better the prospects.—*Journ. A.M.A.*, vol. lxiv, p. 952.

Treatment of Hydrocele by Injections of Formalin.—Soubeyran points out that a large number of substances have been used as injections for the treatment of hydrocele. Solutions of iodine and carbolic acid are most usually employed. The injection of formalin presents considerable advantages. The only instruments required are a small trocar and a hypodermic syringe. The patient lies on his back, and local anæsthesia is unnecessary. The site of puncture is decided by the position of the testicle, the trocar being thrust downwards and outwards when it is posterior, or backwards when it is in the anterior part of the scrotum. The selected spot is painted with tincture of iodine, and the trocar inserted till it can be felt moving freely in the tunica vaginalis. The needle of the hypodermic syringe is then inserted in the same direction slightly above the trocar, and should also be felt moving freely in the cavity. The trocar is then withdrawn to permit the escape of the fluid, care being taken that the sac is not completely emptied. When sufficient fluid has been withdrawn the cannula is removed, and by the needle of the hypodermic syringe 2 to 4 c.c. of a mixture containing equal parts of formalin, alcohol, and glycerine, is injected into the sac. The injection should be made very slowly, the point of the needle being turned about in different directions, so as to ensure the distribution of the solution and its mixture with the remaining hydrocele fluid. The needle is then withdrawn, and the punctures closed with a little collodion, the scrotum being well kneaded to ensure the distribution of the injection.

The operation is but slightly painful, and the patient may be at once allowed to go about. After a few hours some swelling and œdema occur, with a slight increase in the amount of fluid. This reaction is much less marked than after the injection of iodine, and commences to subside in four or five days, and should be complete in about a fortnight. This method is much less painful than the iodine injection, both at the time of operation and during the reaction, and avoids the risks of iodism, while as regards freedom from relapse it is at least equal to the older method.—*Journal des Praticiens*, p. 17.

THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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THE IMPORTANCE OF NEW SYMPTOMS IN OLD-STANDING DISEASE.*

By G. GREY TURNER, F.R.C.S.Eng., Major R.A.M.C. (T.F.),

Assistant Surgeon Royal Infirmary, Newcastle-on-Tyne, Lecturer on Surgery, University of Durham College of Medicine.

THE patient you see before you is a man, æt. 59 years, who came to the Out-patient Department two months ago complaining of a large right inguinal hernia which had been present for ten years. He had always been able to reduce it and had never worn a truss, but during the previous three months it had increased in size and had given him more trouble than previously. Some of you may remember that, considering the man's age and the fact that the hernia had only recently commenced to annoy him, we examined him very carefully, with the especial object of discovering the existence of some other condition which might have directed his attention to the hernia, but with a negative result. As the man appeared to be a good subject, with healthy arteries and urine, we determined to perform a radical operation in spite of his age, and he was accordingly placed on the waiting list for admission. In the ordinary course of events this patient would have been sent for and operated upon, but owing to great pressure on our beds this had not been done, when he re-applied to the hospital for treatment on account of increase in size of the abdomen, supposed to be due to some obstruction in the hernia. You will now see that the abdomen is very largely distended, and that the man has obviously lost flesh, while his skin has a yellowish tinge. On the right side is the large

* A Clinical Lecture delivered at the Royal Victoria Infirmary, Newcastle-on-Tyne.

inguinal hernia which originally brought him to hospital, but which is quite easily reducible, though the sac immediately fills again when released from the pressure of the hand. On searching for an explanation of the distension we find that the abdomen contains a large amount of fluid, and a careful examination and consideration of all the circumstances leads us to the conclusion that the patient is undoubtedly suffering from cirrhosis of the liver, with ascites as the main symptom, while the history of his habits supports this diagnosis.

This case, then, is an admirable illustration of what we frequently observe in dealing with large numbers of patients. With the help of our House Surgeon, Mr. Dixon Johnston, I have tried to put the matter in the form of an aphorism as follows: "The occurrence of unaccustomed symptoms in chronic conditions is suggestive of some new disease."

Quite early in my career my attention was drawn to this subject by a case which impressed me so much that it has never been forgotten. A poor woman who had borne many children came to the hospital complaining of symptoms which she attributed to a rupture of the perineum. Her troubles were referred to the rectum, and on questioning her it turned out that the perineum had been ruptured for many years, and that it was only during the preceding few months that she had thought it anything more than an inconvenience. As nothing further could be made out to explain her symptoms* the perineum was repaired, thus restoring the anal sphincter, and it was expected that the patient would have no further trouble. Within a few months she returned complaining that her symptoms were worse than before; that she had great difficulty in getting the bowels moved; and that she now passed blood and mucus. There was marked loss of flesh, and a growth could be felt high up in the rectum. It was then quite clear that all the time the patient had been suffering from a malignant growth of the bowel. At the operation, which Mr. Morison carried out, this growth was removed, together with the uterus to which it was fixed, and I show you the specimen (Fig. 1), which is a constant reminder to me of the importance of the subject we are now discussing.

* It was before the days of the electric sigmoidoscope (which I first used in the Old Infirmary in October, 1904), bismuth enemata, and X-rays.

Quite a large proportion of the cases in which this matter crops up are in association with hernia, and the next notable instance that I can recall was that of a man who came to the hospital complaining of a large hernia on the left side which had been present for years, but which had only recently given rise to trouble in the form of increasing constipation. He was a healthy-looking old man without any urgent symptoms, but the large scrotal hernia which he presented was irreducible and felt as though the bowel which it contained was crammed full of fæces. We looked upon it as an example of obstructed hernia, and decided to operate. The sac contained the greater part of the sigmoid full of fæcal contents, and with very thick walls, but after dividing the neck it was observed that the part of the sigmoid within the abdomen was also much thickened. This at once aroused a suspicion of obstruction lower down, and on examining the rectum a malignant growth was found which was undoubtedly the explanation of the trouble.

The recital of this case sounds as though an absolutely inexcusable blunder had been made, but it is very curious how these patients are themselves misled, for this man at that time made no complaint of symptoms directly referable to the growth in the rectum, or that could not be explained by the theory of an obstructed hernia. It also furnishes a commentary on the necessity for thorough routine examination, which is a subject we might profitably discuss at some other time.

Not long afterwards another case came under my notice in which a surgeon had performed a radical cure for a long-standing hernia without relief to the patient's symptoms, and when the man returned to the hospital some three months later a carcinoma high up in the rectum was found.

Of course there are circumstances under which even the most careful examination will fail to reveal the presence of some early disease, which, though it may make the patient feel that something is wrong, yet does not give rise to symptoms or signs by which it can be detected. This was so in the case of a man, æt. 44 years, who had suffered from hernia for twenty years, and who was led to seek advice because of a general failure of health, and of some abdominal pain which he attributed to the presence of the rupture. A radical cure was carried out, and apparently with the greatest possible relief, but after a short time his symptoms returned, and

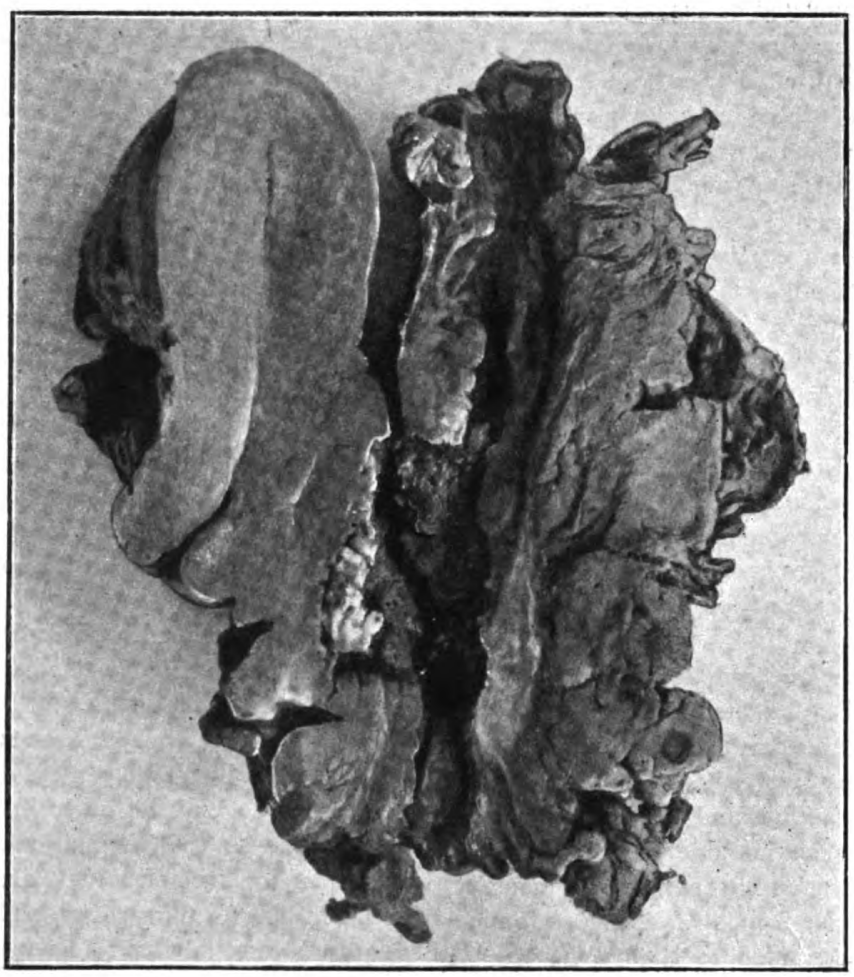

FIG. 1.—Carcinoma of the pelvic colon involving the uterus.

pain in the upper part of the abdomen became very severe. When I again saw him I was shocked at the loss of flesh, and though I suspected malignant disease of the stomach I could find no definite signs. Soon afterwards he developed jaundice and some hard, enlarged glands in the left posterior triangle, definitely settling the diagnosis of malignant disease, and heralding his death, which occurred a few weeks later.

Again the symptoms may be of a more acute nature, as in a recent example in which a man, æt. 62 years, consulted his doctor because of repeated attacks of severe cramping pain in the abdomen which he attributed to an old-standing rupture. The doctor found that the hernia was reducible and ordered the patient a truss, which appeared to be satisfactory; the man experiencing considerable relief. Within a very short time the symptoms recurred, and I was asked to go down and operate for strangulated hernia. Not finding the hernia completely irreducible, and with the story that it had been present for such a number of years while the attacks had only been comparatively recent, I became suspicious that the patient was really suffering from a bowel growth, and measures were taken to establish this diagnosis. An enema was given, which not only failed to give the patient relief, but made matters worse, and when seen a few hours later the cæcum could be distinctly felt distended and contracting. At the operation a malignant growth was found in the transverse colon, explaining the whole of the circumstances of the case.

Last summer I was asked late one night to go a

long journey into the country prepared to operate on a case of strangulated hernia. I found an old gentleman, æt. 72 years, obviously very ill. His symptoms were attributed to a large left inguinal hernia which had been present for many years. He had severe abdominal pain and vomiting, and had not passed flatus since the onset of his illness in the early morning. To make a long story short I explored the hernia, only to find the signs of peritonitis with free fluid and gas, which a second incision in the upper abdomen showed to be due to a ruptured duodenal ulcer.

Elderly men who are developing enlarged prostates are sometimes apt to attribute the increased difficulty in micturition to the presence of a hernia, usually of long standing. I can remember more than one such case in which an operation directed to the cure of the hernia ought more properly to have been an operation for the removal of the prostate.

All surgeons are familiar with the small epigastric herniæ about which patients often so bitterly complain, and which are so frequently associated with the neurotic state. Nevertheless in these cases it behoves us to be cautious and to carefully examine the patient, lest some condition more serious is attributed to the innocent hernia. A man, æt. about 32 years, complained of severe abdominal pain which he attributed to such a hernia. A careful examination conducted before a class, with a view of eliminating any more serious condition, failed to discover sufficient reason for the symptoms, and rather yielding to the importunity of the patient I performed a radical cure for his small, but very definite, epigastric rupture. Not long afterwards the doctor informed me that the patient was worse than before and was losing flesh. When I had the opportunity of making another examination I found a large mass occupying the epigastrium which was probably a retro-peritoneal growth, for the man died soon afterwards.

The same sort of thing is particularly apt to occur in women suffering from umbilical herniæ. As you know, this particular type of rupture is very liable to become strangulated, sometimes in quite a mild way, producing attacks of abdominal colic, and so accustomed do these women become to these attacks that any pain from which they suffer is usually credited to the obvious external trouble. Not long ago such a patient was sent to the hospital as an example of strangulated umbilical hernia, but an examination did not disclose any of the local signs associated with trouble in the hernia, and the patient was therefore kept under careful observation. Next day she was a little jaundiced, and we were able to feel a distended gall-bladder, which was dealt with at a subsequent operation, many gall-stones being removed.

Some of the most striking instances in illustration of the text we have chosen arise in connection with hæmorrhoids. I am sorry to say that scarcely a month passes without my seeing some case of more or less serious rectal mischief which has been for too long attributed to old-standing piles. Some of you may remember the case of a man in Ward 5, whose rectum was excised for carcinoma, and who told us that for two years he had been complaining of symptoms which were attributed to hæmorrhoids, and that it was while introducing a suppository which had been ordered for his relief that he himself first felt the hard growth which turned out to be malignant. It is very pathetic to think how much it would have been to the advantage of this patient if a timely rectal examination had been made. Only the other week a man, æt. 54 years, came to the hospital asking to have an operation for hæmorrhoids. He said they had bothered him for thirty years, and though he had frequently been advised to have them removed it was only the accession of new symptoms that had made him consent. In that case a piece of the growth was actually invading the anus, and projected just like a pile, as you will see by reference to the specimen (Fig. 2). You will observe that only part of the growth is shown, but that introduces another matter with which we may deal some other time.

Of course, the cases just quoted are inexcusable ones in which rectal examination would at once have shown the real nature of the trouble, but there are many that are more difficult, and in which possibly a malignant growth may exist high up, and may only be seen with the sigmoidoscope or be discovered after exploratory operation.

I can assure you that there are no cases in which it is more necessary that you should be particularly careful to be sure of your ground than long-standing cases of hæmorrhoids which have only recently become troublesome, and to which patients are only too ready to attribute their troubles.

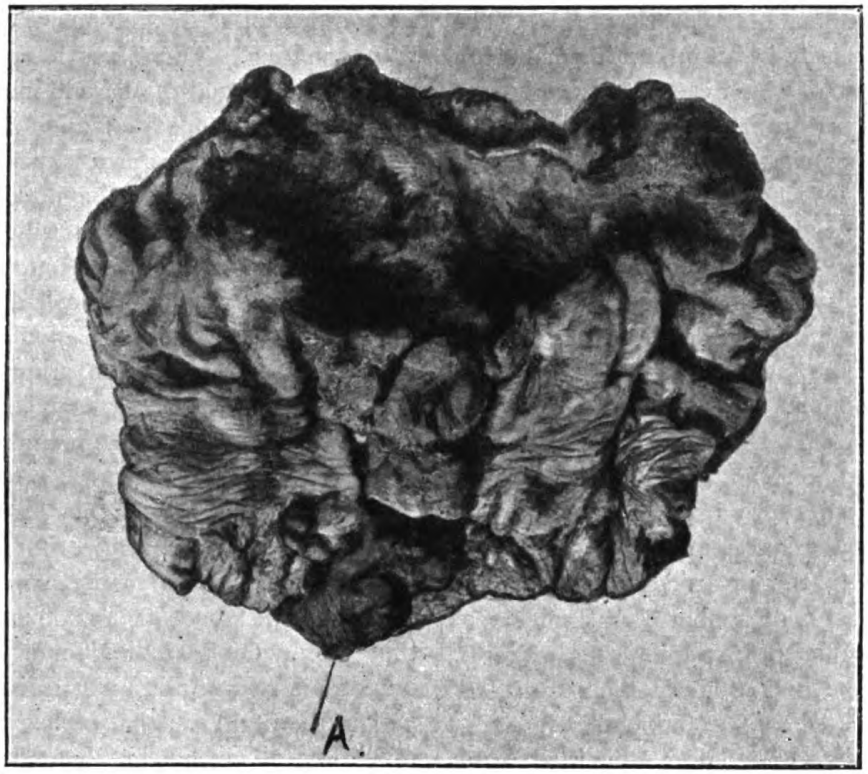

FIG. 2.—Carcinoma of the rectum associated with long-standing piles. A process of the growth is projecting from the anus (A).

The same thing applies to varicose veins, for they are so obvious that the patient naturally blames them for any sort of ache or pain that may affect the limbs. I can well remember that the importance of this matter was insisted on by Sir William Bennett in one of his lectures published in the CLINICAL JOURNAL the year that I qualified.* In such cases you must be on the look-out for peripheral neuritis, the lightning pains of locomotor ataxia, osteitis, new growth, pelvic tumours, and other troubles. I can just find space for one illustration: that of a middle-aged man, who was sent to hospital with a view to the removal of some long-standing varicose veins, which he said had been much more troublesome of late. He certainly had very bad varices in both limbs, but we were struck by his blueness and bloated appearance. This led us to make a more extended examination, when we found that he was suffering from mitral regurgitation, that there was albumen in the urine, and that he confessed to being addicted to alcohol. Of course, the idea of operation was scouted, and under proper treatment he greatly improved, and was able to return to his work, at all events for a time.

Enlargements of the thyroid, in the past at all events, were very commonly looked upon as of no moment, and especially so among people living in so-called goitrous districts. I have, however, seen so many cases in which malignant disease has developed in connection with a long-standing goitre that I am always very careful in examining cases in which there is a history of new symptoms or exacerbation of long-standing trouble. In November of 1911, a man, æt. 63 years, sought advice about an enlargement of the thyroid. He said that he first noticed the tumour ten years previously, and that it had always given him some trouble in breathing,

* "A Lecture on Some Points connected with the Treatment of Varicose Veins of the Lower Limbs" (THE CLINICAL JOURNAL, January 19th, 1898, vol. xi, No. 13).

but that since August the swelling had rapidly increased in size, and had produced not only great difficulty in breathing, but also dysphagia. The condition turned out to be an infiltrating malignant growth of the thyroid, which we were powerless to relieve except by performing tracheotomy.

There is another class of case in which many examples of what I say are to be found, and though they usually come within the domain of the gynæcologist you must clearly understand that there are no sharp divisions in disease, and that the general principles which underlie one branch of medicine are just the same as those that form the foundations of another.

Not very long ago I was asked to see a woman who was known to be suffering from a large fibroid, but who had only recently begun to be troubled with attacks of abdominal pain. She very naturally attributed these to the presence of the obvious tumour, and I must confess that I was unable to find any other sufficient reason for her seizures. Nevertheless, at the operation for the removal of the tumour, I took care to examine the appendix and the gall-bladder. The latter contained many stones and showed definite evidence of recent inflammatory mischief, though the fibroid itself was perfectly innocent. A more serious sequel followed in the case of an old woman, æt. 65 years, who had endured a rectocele for the greater part of a long life. During the preceding few months she had commenced to have more discomfort than ever before and to complain of some abdominal pain, which symptoms she of course attributed to her old complaint, and it was for its cure that she was sent to me by her doctor. From the time of my first seeing her I felt sure that there must be some other explanation, and I fought shy of any interference. However, after keeping her under observation for some time and employing various methods of examination, I was tempted to operate—though I think very foolishly—because I am so familiar with the great relief which these poor people often derive from a proper operation. Nevertheless, I examined the abdomen carefully under the anæsthetic, and I deliberately opened the pouch of Douglas, so that I might explore the peritoneum with the fingers, but without discovering anything to confirm my suspicions. For a short time the patient was greatly relieved and expressed herself as well pleased with the result, but, unfortunately, this relief was only short-lived, for within a few months I was asked to see her again. She had then lost a good deal of flesh and was very much troubled with vomiting. There was a hard infiltration in the neighbourhood of the umbilicus. As she had previously been operated upon for an umbilical hernia, I explored this area with a local anæsthetic, in the hope that it might be nothing worse than a small omental recurrence which had become incarcerated. However, new growth showing the characters of a columnar-celled carcinoma was found, and the patient soon died without anything turning up to determine the primary focus.

The changes that may occur in connection with moles are very characteristic, and one has seen so many examples of melanotic sarcoma developing that a consideration of this group alone would form quite a profitable subject for a clinical lecture. A few months ago a man, æt. 51 years, was sent to consult me about a mass of enlarged glands in the groin. On searching for a possible focus I observed a scar on the inner side of the leg. The patient then told me that he had had a pigmented mole there all his life, but that two years previously it had become ulcerated and refused to heal and was excised. I subsequently ascertained from the surgeon who operated that the "mole" had become a melanotic sarcoma. The man developed enlarged glands in various situations, lost flesh rapidly, and eventually died just three years after he first noticed any change in his birth-mark.

Secondary changes are apt to arise in connection with all sorts of external tumours, but often the patients have become so accustomed to the presence of a swelling that they neglect important changes, and by the time they seek advice the superadded disease is often too far advanced to be dealt with successfully. This was well illustrated in the case of a man who was sent to consult me about a parotid tumour. The doctor's letter stated that: "The tumour has been present for twenty years, but recently it has grown larger and within the last fortnight has had some septic infection added." I found an infiltrating malignant growth, deeply adherent and with many enlarged glands, in the neck. The man was astonished when I had to inform him that the condition was already beyond the hope of successful removal.

Similarly there are women who harbour lumps—tumours—in their breasts for years and who never

consult a doctor until there is such a marked change as to mean that cancer has supervened. Unfortunately, in its early stages cancer is not the painful disease the public expect; if it were so cases would come to the surgeon earlier, and untold suffering would be avoided.

So far we have dealt with conditions in which there has been some obvious external manifestation likely to mislead the patient and often the medical attendant, but you must be familiar from your experience in the medical wards with many internal conditions in which the same care is necessary lest some new disease should be missed under the guise of an old malady. I was forcibly reminded of this by the circumstances of a recent case in which a man, æt. 58, with irremovable cancer of the stomach, gave the following history:

For many years he had suffered from indigestion. The last few months he had become much worse, but had grown so accustomed to pain that he thought nothing of it. It was only a few days before my seeing him that he went to his doctor, who discovered a big hard mass in the epigastrium.

Then there is still another group of cases in which you must be very careful, namely, those in which some external and obvious trouble is blamed for a general failure of health which at first sight is not likely. Quite recently an elderly woman was admitted to the Infirmary for the radical cure of a large umbilical hernia. On going carefully into her history, it appeared that she was really troubled by a general loss of health and strength which naturally enough she had attributed to the rupture. An examination of the urine showed it to be full of sugar, and observation over a considerable time confirmed the diagnosis of diabetes rather than a temporary glycosuria, such as is not infrequently found in elderly women of the sort likely to suffer from umbilical hernia. Another case of the same kind was that of a young lady, who came to the out-patient department with the request that we should remove some small, hard, discoloured tumours which were present in connection with the skin along the inner side of the left foot. The diagnosis was interesting, and finally we concluded that they were probably thrombosed cavernous nævi. We were struck by the fact that though the patient had noticed the lumps for a very long time she had only thought of having them removed recently, but she said that as they were affecting her health she was anxious that the operation should be performed. She was thin but bright and vivacious, and on inquiry did not complain of any cough, while the family history was good. The chest was examined and found to be healthy, and I have no doubt that, had we not been anxious to procure a photograph of the foot, the little masses would have been removed by the house-surgeon, the patient being allowed to return to her own home the same night. Fortunately this was not done, and a calamity was probably thereby averted, for when she returned on a subsequent occasion my colleague, Mr. Hamilton Drummond, was so struck by her appearance that he procured a sample of her urine and found it full of sugar. This, again, was an example of a young woman who, finding herself not feeling quite in her usual health, was led to blame a condition which had been present for long, and which was really perfectly harmless. A similar instance was presented by a gentleman who had nourished a lipoma of the back for many years, and without its giving him the least anxiety. On finding himself not quite in his usual health, and thinking of some possible explanation, he went to see his doctor for an opinion as to whether the simple tumour should be removed. While the matter was under consideration, the patient passed a little smoky urine, and it was then found that he had other symptoms indicating some type of chronic nephritis which was clearly the reason for his loss of health.

These, then, are but a few instances of what you will all meet with in your daily work, and I hope that their recital will at least encourage you to examine your cases carefully, and avoid hastily coming to conclusions that may be of great importance to your patients.

June 21st, 1915.

Radium Treatment of Cancer of Rectum.—Pellizzari reports a case in which the rectum was almost entirely obstructed by a cancer, about 8 cm. above the anus, and adhesions to neighbouring organs rendered operative treatment too dangerous. The man was given a course of radium treatment in 1913, and again in 1914, and soon regained strength and increased in weight. There is still a slight bulging of the rectum wall at the spot, but the malignant disease has otherwise completely retrogressed.—*Journ. A.M.A.*, lxiv, p. 1538.

CLINICAL JOTTINGS.

(*Continued from p.* 192.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

On the Rupture of Cardiac Valves.

Healthy cardiac valves do not rupture, even with the greatest amount of muscular strain to which they can be subjected. If a valve rupture it is found to be the seat of antecedent mischief, and though the actual rupture may follow some exertion, it may occur spontaneously without any special strain. I suppose a heavy fall or blow might tear a healthy valve just as I have once seen it tear the healthy aorta itself right across as if cut with a knife, but I do not know of any instance of such an effect upon a valve actually recorded.

It is the aortic valves which usually rupture, for they belong more to the aorta than to the heart, and are readily involved in those degenerative changes which specially affect the aorta, viz. atheroma and syphilis. Sometimes the valve is torn across; sometimes the edge remains unbroken and the belly of the valve is perforated, either with or without the previous formation of a small aneurysm in it.

Ulcerative endocarditis, or that form of malignant endocarditis in which there is much erosion of the valve, may also lead to tearing or rupture, but these cases fall clinically into a different category, and the symptoms are those of the original disease, viz. septic infection in which more or less definite, but often very indefinite, cardiac signs develop.

In ulcerative endocarditis the aortic valves usually show most advanced destructive lesions, yet the mitral valves may do so too, especially when the chordæ tendineæ are broken. With this exception the mitral valves are rarely ruptured; but now and then when a tag hangs from the aortic valves, which, as it flaps to and fro, strikes the aortic cusp of the mitral valve, it may rub a spot there which may ultimately give way, or lead to an aneurysm there projecting on the auricular surface, with a perforation at its tip.

The evidence of rupture is given by the sudden onset of cardiac symptoms, viz. pain, dyspnœa, and cardiac embarrassment. These symptoms may set in suddenly in a patient who has been apparently well till the onset, though post-mortem evidence shows that the valves have been long unsound. In other cases the patient is known by the physical examination to have had valvular lesions, though probably without marked cardiac symptoms prior to the rupture.

On the Tapping of Hydatids in the Liver or Abdomen.

A hydatid may be tapped with safety so long as it is within the liver, *i. e.* so long as it is shut off completely by liver substance from the cavity of the abdomen. If the hydatid be within the abdominal cavity, whether originating in the liver or not, tapping is a very risky procedure, for then either the daughter-cysts, if there be any, make their way out from the puncture into the peritoneal cavity, where they may grow, and so the last state of that patient be worse than the first; or what is more likely, the cyst becomes infected from the intestines, septic symptoms arise, and the abdomen has to be opened urgently under very unfavourable conditions.

Nowadays, if a hydatid has been diagnosed, the abdomen is opened as a matter of course and the cysts dealt with as circumstances suggest. Paracentesis is now hardly ever done except for diagnosis.

What has been said shows when exploratory puncture is justifiable for diagnosis and when not. Except when it is certain that the hydatid is deeply seated in the substance of the liver it should not be tapped. Even then paracentesis can only cure the simple cyst, and there are no means of determining beforehand with certainty whether the cyst is, or is not, proliferating.

(*To be continued.*)

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DIAGNOSIS AND TREATMENT OF ILEAL STASIS.*

By CHALMERS WATSON, M.D., F.R.C.P.E.,
Assistant Physician, Royal Infirmary, Edinburgh; Lecturer in Clinical Medicine, Edinburgh University.

GENTLEMEN,—I propose to draw your attention to-day to the subject of ileal stasis, and to illustrate its clinical features by showing you two cases at present under my care in the ward. It will enable us to see this subject in its proper perspective, if at the outset I give you a brief survey of the great development in abdominal surgery that has occurred in the past twenty-five years. That period of time may be roughly divided into four eras.

(1) *Acute appendicitis.*—Twenty-five years ago, in my student days, appendicitis or, as it was then often called, perityphlitis was essentially a medical disorder. It was treated by rest, medicinal remedies, local application of cold, and other medical measures. Between the years 1890 and 1895 this condition came to be recognised as one that was more appropriately treated by the surgeon. It is probable that about this period and subsequently there was a very great increase in the incidence of appendicitis. Certainly the great increase in the incidence of this affection appeared to be far in excess of what could be reasonably explained by increased skill in diagnosis. It is probable that one of the causes of this increase has been the modifications in food and feeding that have occurred in this period, the more important modifications in all probability being a greatly increased consumption of sweets of all kinds, and the use of preservatives in the manufacture and preservation of foods.

(2) *Gastric surgery.*—The next important development of surgery was the application of surgery to the treatment of gastric and duodenal ulcers

* Lecture at the Royal Infirmary, Edinburgh.

chiefly by the operation of gastro-enterostomy. This operation was in great vogue for several years, and in course of time statistical evidence became available as to its value in the treatment of cases that were clinically diagnosed as gastric ulcer. As years passed, and the after history of cases was ascertained, it became increasingly apparent that a large number of cases, clinically diagnosed as gastric ulcer and treated by gastro-enterostomy, derived little or no benefit from that operation, and the operation is at the present time much less frequently performed. Attention now came to be directed to the appendix as a cause of gastric disorders.

(3) *Chronic disease of the appendix.*—The next development was the recognition of the fact—largely owing to the writings of Paterson, Mayo, and Moynihan—that many disorders of the stomach, associated with symptoms which very closely resembled gastric ulcer, were dependent upon a chronic catarrhal lesion in the appendix, so-called appendicular indigestion or appendicular gastralgia, complete relief being afforded by removal of the appendix. As a consequence, the view, so well expressed in the following epigram, came to be widely held: "The most frequent seat of gastric ulcer is in the right iliac fossa." The diagnosis of appendicular gastralgia now became a very fashionable one, and operation for the removal of the appendix was performed in great numbers, often, it is to be feared, on rather slender grounds. While marked benefit followed the operation in many cases, there is no question that large numbers of appendices were removed without any benefit to the patient, with the result that in recent years there has rightly been a reaction against the excess of operations performed in this direction. It is possible that the limited nature of the incision that was largely adopted at this time prevented the recognition of other important lesions which are now recognised in the ileo-cæcal region. This brings me to what may be described as the present era, in which it is becoming increasingly recognised that the *fons et origo* of many gastric and intestinal disorders is to be found in the ileo-cæcal region, associated with not one, but many pathological conditions, of which the subject of my lecture is one of the most important. This era may for convenience be described as one of intestinal stasis.

(4) *Intestinal stasis.*—In recent years the clinical researches of Arbuthnot Lane have done much to advance our knowledge of abdominal disorders, and also to stimulate and direct the attention of clinicians to the necessity of fresh investigation along the lines to which I am drawing your attention to-day. For many years Lane has held the view that disease in the ileo-cæcal region, associated with a pathological stasis in the terminal portion of the ileum, cæcum and colon, is a condition of the greatest importance in inducing not only gastro-intestinal disorders, but wide-spread constitutional affections. He also holds that gastric and duodenal ulcers are not, as a rule, primary disorders, but are secondary to disease in the ileo-cæcal region. The general correctness of Lane's teaching in these directions is now increasingly recognised.

Physiological Considerations.

I wish to draw your attention to some points in physiology which have an important bearing on this subject, dealing first with the *ileo-cæcal valve* or *sphincter*. In recent years the investigations of Barclay Smith, Keith, and Elliot have added considerably to our knowledge of this subject. Their observations strongly support the view that an important neuro-muscular mechanism is involved in regulating the passage of food from the ileum into the cæcum, and at the same time preventing any cæcal regurgitation. These observations justify us in abandoning the use of the term ileo-cæcal valve, and substituting the term ileo-cæcal sphincter. There is good reason for the belief that the normal stimulus for the relaxation of this sphincter is supplied by the introduction of food into the stomach. In the future we must attach more and more importance to factors which modify the relaxation or the contraction of that sphincter.

The second point to which I would direct your attention is the existence of a *physiological ileal stasis*. I will show you on the screen slides illustrating, in a healthy subject, the arrival of part of a bismuth meal into the terminal coils of the ileum and cæcum, one hour after ingestion of the meal; this rate of movement being, however, above the average. The retention of a bismuth meal in the terminal coils of the ileum for a few hours is a physiological process, digestion and absorption taking place actively from the ileum during this time. The existence of a pathological ileal stasis can be established only by an X-ray examination carried out from five to six hours after the

ingestion of the meal. It is important to remember that this examination should be carried out both in the erect and recumbent postures. As Jordan has pointed out, the latter method yields information of the greatest value, as thereby the parts in the ileo-cæcal region can be differentiated by handling under the screen, which allows the condition of the terminal coils of the ileum to be more satisfactorily determined. The recumbent posture is also necessary for demonstrating effectively the condition of the duodenum, which Jordan has shown to be very frequently dilated in cases of ileal stasis. These points will be demonstrated to you by means of lantern slides.

The third point to be noted is *the relative sterility* of the contents of the ileum under normal conditions, this contrasting with the abundant bacterial flora on the distal side of the ileo-cæcal sphincter. I think it probable that a degree of incompetence of this sphincter, allowing regurgitation of bacteria into the small bowel, is an important factor in inducing disease in this region and subsequent general manifestations of ill-health. I show you here a post-mortem specimen of the terminal coil of the ileum, cæcum, appendix, and part of the ascending colon, which I have prepared in order to demonstrate the readiness with which fluid introduced into the cæcum overcomes the ileo-cæcal sphincter. While great caution is necessary in drawing any conclusions from such post-mortem material, the information gained from the study of many specimens has indicated to me the importance of further attention being directed to the question of the competence or imcompetence of this sphincter.

Pathological Considerations.

There is no doubt that a pathological ileal stasis is frequently met with. There are two main types: In one there is a marked degree of stasis revealed by the X-rays, which is not dependent upon any mechanical condition of the nature of adhesions, kinks, etc., in the ileo-cæcal region. This variety is termed by Lane "static" stasis. The other, more common, is found in association with adhesions in the ileo-cæcal region, which may be in the form of adhesions passing to the terminal coils of the ileum, from the appendix, or in the form of bands extending from the pelvic brim to the terminal coils of the ileum, which interfere mechanically with the normal functional activity of this part of the bowel. Considerable discussion has arisen as to the exact origin of many of these bands, some authorities regarding them as anatomical structures, others as pathological. The point is one more of academic than practical interest. The practical question which we clinicians have to settle is whether or not any bands, whatever their nature, are interfering mechanically with the functional activity of the part of the bowel affected. I have, in the course of the past few years, seen so many cases which have been ultimately submitted to operation, with resulting permanent benefit to the patient, that I would emphasise to you the importance of paying much attention, not only to the existence of such adhesions, but to a careful study of the cause or causes which have led to their development. I entirely concur with the view which is ably advocated by Adami that a continued latent infection, or, as he has termed it, a "sub-infection" by intestinal bacteria, is an important determining cause of such products of inflammation. It is further important to bear in mind the frequency with which ileal stasis is associated with varying degrees of cæcal and colon stasis. The frequency with which an abnormal condition of the duodenum is found in association with ileal stasis has already been referred to. My experience has amply confirmed Jordan's views as to the frequency of dilatation of the duodenum, with increased elongation of its second part, and the occurrence of reverse peristalsis in the duodenum in cases of marked ileal stasis.

The remaining pathological point to which I wish to direct your attention is the bacteriology. A pathological ileal stasis is probably invariably accompanied by an abnormal bacterial activity in the ileum. The ileal contents are under normal conditions relatively sterile, but in the cases under consideration a bacteriological examination has repeatedly shown a very abundant bacterial flora, the bacteria being chiefly of the coli group, which have doubtless come from the cæcum. As I have already indicated it is probable that a regurgitation from the cæcum, which allows of an ascending infection of the bowel by bacteria of a low degree of virulence, is an important factor in developing intestinal toxæmia. In this connection it is interesting to note that I have lately had the opportunity of making observations on the state of the stools in two patients

after a complete and very successful—from the surgeon's point of view—colectomy. I found in both instances that the stools passed by the patients, alike in their consistency, odour, and bacterial contents, did not differ materially from that passed by an unhealthy large bowel. It was clear that in both cases the condition of the lower part of the small intestine was identical with that seen in aggravated cases of excessive putrefaction in the large bowel. In other words, in advanced degrees of intestinal toxæmia the lower part of the ileum may play the part of a cesspool analogous to what more frequently obtains in the large bowel.

Let us now look at the clinical aspect of the subject.

Clinical Conditions associated with and dependent upon a Primary Ileal Stasis.

These will be very briefly mentioned, and may be considered under the following groups. An illustrative series of slides under each group, taken from cases that have been under my observation in the past few years, will be demonstrated, the slides being taken in each instance from cases that have been submitted to operation, in which the after-history is known.

(*a*) *Functional disorders, so-called*, including conditions of indigestion and neurasthenia. This includes a very large and important group of disorders.

(*b*) *Duodenal or gastric ulcer.*—There is good reason to believe that these disorders, and more particularly duodenal ulcer, are very frequently, as Lane contended, a late sequel of disease in the ileo-cæcal region. The facts recently recorded by Wilkie are important in this connection. In a consecutive series of 490 cases which he examined in the post-mortem department of the Royal Infirmary recently, a duodenal ulcer was present in 41, or nearly 10 per cent. of the total. In 21 cases there was one ulcer, in 16 two ulcers, and in 4 more than two ulcers. In only 6 cases a diagnosis of duodenal ulcer had been made during life, the patients having died from some other disorder. Wilkie found in the majority of these cases a definite pathological lesion in the ileo-cæcal region, involving the ileum, cæcum or appendix, his observations leading him to the general conclusion that disease in this region is a primary factor in the development of duodenal ulcer.

(*c*) *Arthritic disease.*—There is little doubt that many forms of chronic joint disorders now classified under the vague term of rheumatism, or rheumatoid arthritis, are a direct result of septic absorption in subjects, in whom ileal stasis, associated with cæcal or colon stasis, is the most important pathological condition in the early stages of the disease. *It is important to bear in mind that all sorts of vicious circles become established, so that we must not restrict our attention to any one part of the digestive tract, but study carefully the functional activity of the whole prima via.*

Before referring to the question of treatment, reference must be made to accurate diagnosis. In addition to a careful study of the history and the examination of the abdomen by ordinary methods, reference may be made to the special value of the X-rays already referred to; also to the importance of repeated examination of the stools in order to ascertain the state of the large bowel, and to the advisability of a careful examination of the urine, including in many instances a bacteriological examination. The more important urinary abnormalities met with include: (*a*) The presence in excess of cellular elements, especially epithelial cells and pus cells; (*b*) an increased toxicity of the urine revealed by the abnormal multiplication of bacteria after voiding, and in many instances a true bacteriuria, chiefly organisms of the *B. coli* and coccal groups; (*c*) the presence of oxalate of lime crystals; and (*d*) excess of indican.

I will now demonstrate the following two cases:

(1) David S—, æt. 43 years, a clerk, was admitted to my ward complaining of severe pain in the epigastric region usually commencing in the evening, with increasing asthenia of about ten years' duration. His illness started acutely about ten years previously with severe abdominal pain and vomiting; he was in bed for two months at this time, and he had had recurring attacks of pain with obvious gastric disturbances since. The pain usually came on about 8 o'clock in the evening shortly after supper, always localised to the epigastric region, often severe, and keeping him awake for most of the night. It was relieved by hot applications. He used to vomit about twice a week, usually about 11.30 p.m., but the vomiting

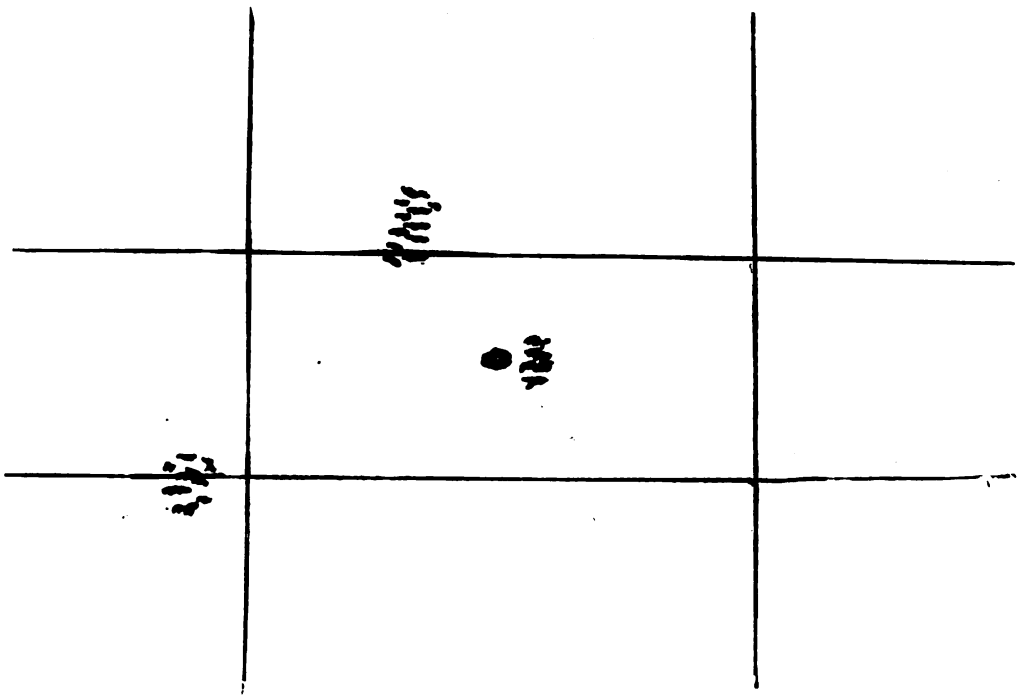

Diagram illustrating the areas of tenderness in D. S—. These areas are often met with in cases of marked ileal stasis.

has been less frequent of late. His appetite has been fair, but he is much troubled with flatulence and water-brash. During the last few years he has frequently been in bed for a week or two at a time. His weight is about a stone below his normal. He has suffered from constipation since the onset of his illness ten years ago, and has been much troubled with headache. There is nothing to note with regard to previous illness, personal habits or heredity.

The general condition of the patient is very unsatisfactory. His face is thin and drawn, his nutrition very poor, and his complexion pale and sallow. On objective examination his tongue is flabby and dirty, his breath slightly offensive, and there is a considerable degree of pyorrhœa. On examination of the abdomen there is distinct pain on pressure in the right iliac region, also tenderness on pressure immediately to the left of the umbilicus and over the upper part of the right rectus muscle, which shows a distinct degree of rigidity (see Fig.). There is marked gurgling in the ileo-cæcal region. On examination by a test meal the acidity of the gastric contents was found to be normal. On examination after a bismuth test meal the stomach was found normal in position and action. After six hours there was an abnormal retention of the bismuth meal in the terminal coils of the ileum, and after forty-eight hours a moderate degree of colon stasis commencing at the hepatic flexure. The motions were constipated and offensive. The pulse was weak, and the heart sounds feeble. This combination of symptoms and physical signs points clearly to a primary lesion in the ileo-cæcal region, interfering with the free passage of the ileal contents into the cæcum, probably due to adhesions, which may be secondary to old standing appendix mischief. In view of the chronic history and the local condition it is inadvisable to waste time over a prolonged course of medical treatment, and it has been arranged, therefore, that an operation be performed.

At the operation, which was performed subsequently by Professor Thomson, the correctness of the diagnosis was established. Definite kinking of the ileum was found about four inches from the termination of the ileum, due to adhesions from the pelvic brim. The appendix was acutely kinked in its middle, and tied down by adhesions apparently resulting from old standing inflammation. Other viscera appeared to be normal, with the exception of the first part of the duodenum, which was slightly

dilated. The adhesions were removed and the appendix excised. There is little doubt that a satisfactory return to good health will be attained by the operation, and that this will be maintained, especially if steps are taken by suitable exercises and massage to improve the weakened tone of the intestinal and abdominal musculature.

(2) The second case is that of Mary R—, æt. 28 years, single. She was admitted complaining of pain in the stomach, vomiting, and general weakness of ten years' duration. The illness began with pain in the region of the stomach, this being at first of an intermittent kind; it might last for a few hours or a whole day. At first there would be intervals of freedom for a month or more, but these intervals gradually diminished in length until in the last year there was seldom more than a day or two's freedom from pain. Her illness has been accompanied by increasing disability for house work. There is no history of hæmatemesis, or melæna, and she is not much troubled with wind or water-brash. The patient thinks she is rather better on a full diet than on a restricted one. She has been in bed for weeks at a time on account of her stomach trouble. She has had no previous illnesses of any consequence, but has never been robust. Her personal habits have been satisfactory; her mother was a martyr to indigestion.

The general condition of the patient is poor. She is anæmic, thin, with a soft flabby muscular system. With regard to her digestive system the patient complains of pain in the umbilical region, passing through to the back, and sometimes to the left side, usually fairly severe, and lasting many days, which is relieved by vomiting, the vomited matter being usually sour fluid, and occasionally bile. The pain is not notably influenced by the ingestion of food. The bowels are stated to be regular. Objectively, the tongue is flabby and furred, the teeth are all artificial, the teeth having been in an unsatisfactory state for a long time prior to removal a few years ago. The abdomen is exceedingly soft and flabby with marked splashing in the ileo-cæcal region, and also to a much less extent in the epigastric and left lumbar region. On X-ray examination very marked ileal stasis was present, with a nearly equally pronounced general colon stasis, the whole colon containing bismuth after forty-eight hours. There is no evidence of disease of the gall-bladder. Other systems are apparently normal with the exception of: (1) A weak condition of the circulation generally; (2) numerous catarrhal cells in the urine, and (3) slight excess of indican. The combination of the history with the clinical condition described point clearly to the ileo-cæcal region being the primary source of the mischief, the ileal stasis being, in all probability, the primary factor. It is probable that this depends upon a local mechanical condition which can be dealt with effectively in the first instance only by a surgeon, and accordingly the patient has arranged to come back to hospital shortly for operation.

Treatment.

It will be convenient to discuss this under the two headings of medical measures and surgical treatment. In the earlier stages of the disease, ileal stasis, and the associated stasis commonly met with in the cæcum and colon, is essentially a medical ailment and can be corrected by appropriate treatment. In chronic cases accompanied by pathological adhesions involving the ileum, and other evidences of disease involving the cæcum and appendix, the condition can be dealt with satisfactorily only by surgical measures. It is sometimes a matter of considerable difficulty to determine in any given case whether the case can be dealt with successfully medically, or can be relieved only by surgical measures. The important points in this connection are: (*a*) The history with regard to chronicity and the amount of disability entailed; (*b*) the result of medical treatment, if that has been thoroughly applied; (*c*) the information supplied by the X-rays.

Medical measures.—The various medical measures which are called for in any given case depend to a large degree on the extent to which the large bowel is affected in conjunction with the terminal portion of the ileum. Then again, the question of whether medical treatment involves the patient being laid up in bed depends much more on his general condition than on the local condition of the bowel. As a general rule, it may be said that in all cases where the general health is notably impaired, as shown by the existence of neurasthenic symptoms, loss of weight, anæmic condition of the blood with feeble circulation, complete rest in bed for a few weeks is advisable.

The medical measures may be discussed in the following order.

(1) *Remove any contributory cause of sepsis*, such as is frequently present in the shape of a neglected pyorrhœa. Long standing oral sepsis is an important factor in inducing or aggravating the unhealthy septic state of the digestive tract which is sometimes the primary factor in the development of the condition.

(2) *Massage and remedial exercises.*—This is by far the most important remedial measure in the treatment of the disease. The underlying defects are the atony of the intestinal muscles, a want of nerve tone in the sympathetic nervous system, and a venous stasis in the abdominal circulation, all of which can be improved by the judicious and skilful use of massage and remedial exercises. It is essential, however, that a definite prescription for the massage and exercises be given by the practitioner, and it is important for you to avoid the common error of handing these cases over indiscriminately for treatment to a masseur. It is important to keep in mind the following points in connection with the prescription: (*a*) The value of manipulative movements of the spinal column directed to improve the circulation in the cord, and through that to influence the whole nervous supply of the abdomen; (*b*) the importance of exercises directed to increase the functional activity of the liver, which is so largely concerned in neutralising the toxins which are continuously being absorbed from the digestive tract; and (*c*) the importance of avoiding excessive massage to the abdomen. It is seldom advisable to massage the abdomen longer than from ten to fifteen minutes. The best results are got from a judicious combination of massage and remedial exercises such as I have described to you and illustrated with the assistance of a masseur in a former clinical lecture. Such treatment has, as a rule, to be continued for at least four to six weeks if any permanently favourable result is to be attained, and during this period it is inadvisable for any purgative medicine to be taken. Supplementary measures in this connection are the administration of a tumbler of hot water in the morning, and a morning visit to the closet, regardless of the absence of any inclination.

(3) *Intestinal lavage.*—In cases where a careful examination of the stools reveals pronounced excessive putrefaction, it is often advisable at the outset of treatment to treat the lower bowel by giving an enema of soap and water daily, followed immediately by irrigation of from two to three pints of clear water. This may effect a material improvement in the condition of the colon and allow the subsequent use of the massage and remedial exercises to exert their favourable effects. Care must, however, be exercised in the prolonged use of intestinal lavage, as in some instances its continued use appears to excite a reverse peristalsis of the upper part of the colon, which aggravates the absorption of poisonous material from the bowel.

(4) *Diet.*—The dietetic treatment should be carried out on general lines. Speaking generally the diet is that applicable to constipation and should include the administration of hot water on an empty stomach at least twice a day, the administration of a reasonable amount of fruit, fresh or cooked, with careful restriction of the amount of sugar taken with it, the administration of brown bread, green vegetables, and oatmeal. Where the state of the digestion permits it is advisable to increase the amount of fat in the form of bacon, butter, and cream, as the lubricating effects of this addition are advantageous. In cases associated with obvious atony and slight dilatation of the stomach the meals should be largely dry, fluids being given half to one hour before meals. In many cases associated with marked flatulence it is advisable to put the patient on a largely protein dietary, especially restricting for a time sweets and farinaceous foods.

(5) *Medicinal.*—In cases associated with hyperacidity the administration of an alkali in the form of 20 gr. of magnesium carbonate and sodium carbonate, three times a day one hour after food, is often helpful. The internal use of petroleum is sometimes beneficial, a dessert-spoonful to a tablespoonful being taken three times a day after food. The beneficial effects of petroleum are not usually obtained until the unchanged petroleum is visible in the stool. In every case where an attempt is made to correct constipation by massage and remedial exercises all aperient medicines should be avoided after the preliminary clearing out of the bowel.

In many cases medicinal remedies can be dispensed with entirely. Slides will be shown to you illustrating the satisfactory results obtained in appropriate cases without recourse to drugs of

any kind. A thorough course of treatment along these lines for a period of from four to six weeks will show clearly whether medical measures alone are going to yield a satisfactory result. If the result is not satisfactory operation should be resorted to. The briefest reference will be made to this aspect of the subject.

The various operative measures that are indicated include the following. It will be seen that these apply not only to ileal stasis, but to the associated conditions of the cæcum and colon which frequently co-exist:

(1) The removal of adhesions involving the ileum or cæcum.

(2) A narrowing and mobilising of the cæcum.

(3) The removal of the appendix.

(4) A combination of the foregoing.

(5) A short-circuiting operation—ileo-sigmoidoscopy or colectomy, partial or complete.

After operation it is often advisable to give a course of massage and remedial exercises, in order to improve the atonic condition of the intestinal and abdominal musculature.

June 28th, 1915.

THE VICIOUS CIRCLES OF NEURASTHENIA AND THEIR TREATMENT. By JAMIESON B. HURRY, M.A., M.D.Cantab. London: J. & A. Churchill, 1915. 3*s.* 6*d.* net.

Dr. Hurry has made the study of "the vicious circles" peculiarly his own as shown by his work on "Vicious Circles in Disease" and a most suggestive article on "Obstruction and its Vicious Circles" which appeared recently in our columns (CLINICAL JOURNAL, May 5th, p. 145). In applying the method of tracing the influence of a circle in detail throughout a single disorder, to that of the difficult subject of neurasthenia, he has successfully accomplished a difficult task. He defines neurasthenia as "a chronic functional disorder due to exhaustion of the neurons usually associated with impaired ideation." He quotes an excellent definition of fatigue and exhaustion from Verworn. The former term is applied to the phenomena arising from the accumulation of decomposition products, and the latter to the phenomena arising from the consumption of the necessary oxygen and nutrient substances. After discussing the pathology of neurasthenia, circles associated with psychoses, and the vascular, digestive, and other symptoms are considered, as well as those associated with the sense organs. In discussing broadly the lines of treatment for this most protean disorder, rest, massage, exercise, and diversion come first in order. Drugs come next, in accordance with the view taken of their importance. Emphasis is laid on the value of early treatment as soon as the symptoms appear. We commend this book to the profession.

A TEXT-BOOK OF INSANITY AND OTHER MENTAL DISEASES. By CHARLES ARTHUR MERCIER, M.D., F.R.C.P. Second Edition. Enlarged and entirely rewritten. London: George Allen and Unwin, Ltd., 1914. Pp. xx + 348. 7*s.* 6*d.* net.

Mr. C. K. Chesterton, in 'Orthodoxy,' states that the popular definition of a lunatic is a person who has lost his reason, while he, in epigrammatic fashion, defines him as a person who has lost everything except his reason. Be that as it may, the author is to be highly congratulated on the book before us. The chief causes of insanity are stated to be inheritance and stress. The elaboration of the second cause is chiefly due to Dr. Mercier, and the fact should be emphasised. The exact means of transmission is regarded as a "feebleness of resistance" or a "vulnerability" or "delicacy of constitution." The author shows that disorders of conduct and mind constitute the key to the full comprehension of insanity. He also points out the prevalent error of regarding the term insanity as synonymous with unsoundness of mind. The mistake of symptomatic treatment is, it is to be feared, as common in mental as in other phases of practice.

Dr. Mercier states that reviewers will rejoice greatly to find no index to his book, and gives abundant reasons, which, however, are not convincing. The average student for whom the book is intended would doubtless find it convenient to find what he wants at a glance, and an index, even if brief, would have been an advantage. Most medical men meet with some phases of mental disease in the course of their career. We can confidently recommend this book as a most useful guide.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE

ON

THE SKIN ERUPTIONS OF SYPHILIS AND THEIR DIAGNOSIS.*

By H. G. ADAMSON, M.D., F.R.C.P.,
Physician for Diseases of the Skin at St. Bartholomew's Hospital, London.

SKIN eruptions are, in one respect, not among the more important symptoms of syphilis; they do not involve the impairment of any useful or vital function, as do lesions of the eye or ear, or affections of the brain or cord, or of the viscera; they are at most an inconvenience and a source of anxiety and annoyance to the patient.

But skin affections are of very frequent occurrence in syphilis, and they are thus of great significance as evidence of the presence and activity of the disease. Often we are entirely dependent upon our diagnosis of a skin affection as the means of knowing whether a patient has or has not syphilis. Syphilitic eruptions are so varied in appearance, and they may imitate or be imitated by non-syphilitic eruptions, sometimes in so subtle a manner that even those who have many opportunities of seeing all kinds of skin complaint may hesitate to say to which category an eruption belongs. For those who have few opportunities diagnosis must be often extremely difficult.

But a good many difficulties may be smoothed away if one bears in mind the particular non-specific eruptions especially liable to be mistaken for syphilis, and if one appreciates the fact that the, at any rate to the student, almost bewildering

* Delivered at St. Bartholomew's Hospital.

varieties of syphilitic eruption are merely modified forms of two or three characteristic types.

Before considering the features of these eruptions I wish to say a few words as to their significance—why they arise and why they take different forms at different periods of the disease.

Briefly, all these eruptions represent inflammatory reactions the result of the presence in the skin and subcutaneous tissues of the spirochætæ carried there by the blood-stream.

In the primary sore we see an inflammatory reaction against the invasion at the site of entrance. The micro-organisms pass this defence and the infection becomes generalised. The eruptions of the secondary period represent renewed efforts towards defence against the now widely disseminated micro-organisms. They are, in fact, analogous to the "cuti-reactions" which we get in a tuberculous subject by the introduction of tuberculin into the skin. Each efflorescence of a secondary syphilitic eruption is a "cuti-reaction" to the presence of spirochætæ in the skin of a now hypersensitive subject. Presently the body defences get the upper hand, either through increasing immunity or through diminishing virulence of the micro-organisms, and the skin eruptions and other secondary symptoms subside.

The patient may from this time have no further signs of active syphilis; or after few or many years a reawakening of dormant micro-organisms may lead to fresh defensive reactions, known as tertiary syphilis.

Thus we have an explanation of some of the distinctive characters of these eruptions which occur at different periods of the disease. The primary sore is single, being the site of infection; the secondary eruptions are widespread and symmetrically distributed, because the spirochætes are now everywhere in the tissues. The tertiary eruptions are often few and irregularly distributed, because they result from long dormant micro-organisms only here and there awakened to activity.

It is my intention here to pass over the diagnosis of the primary sore and to deal only with the eruptions of the secondary and later stages.

Eruptions of the Secondary Stage.

The two most common eruptions of the secondary stage of syphilis are the *roseola* or *macular syphilide*, and the *lenticular papular syphilide*. All other eruptions of this period have their origin in these two or are modifications of them.

I.

The *roseola* or *macular syphilide* appears at about eight to ten weeks after infection, at first upon the abdomen, then on the back and chest and the sides of the trunk. It consists of rounded patches of a pale pink in the slighter forms to a deeper red in the more marked, and of a red-brown colour from the formation of pigment in the later or fading stage. The patches or macules are generally pretty uniform in size, and spaced at regular intervals so as to make a uniform pattern (Fig. 1). But they vary in size in different cases from a quarter of an inch to an inch in diameter, being usually about the size of a threepenny piece. The edges of the macules are not very sharp, but have a blurred appearance.

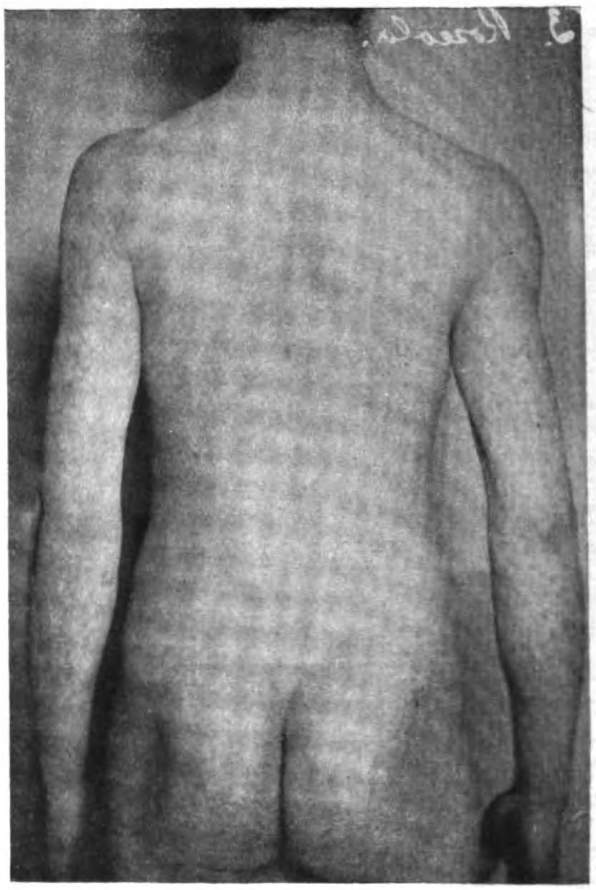

Fig. 1.—Macular syphilide or roseola. The macules are evenly distributed over the trunk, but sometimes also upon the arms and legs, as in the case here shown.

The skin involved does not feel in the least infiltrated, although the microscope does reveal a certain amount of cell-infiltration. More often the eruption is confined to the trunk and upper parts of the limbs, but sometimes the arms and forearms and the legs and thighs are involved, and then the eruption in these parts may be very distinct (Fig. 1) and sometimes slightly scaly. Sometimes—an important sign for diagnosis—there may be a few red or brownish-red patches on the palms. The eruption lasts from a few days to several weeks.

The roseola of syphilis is sometimes so little marked that it fails to attract the patient's attention and is first discovered by the doctor. It is well to remember that it is more difficult to see by artificial light.

Diagnosis.

The macular syphilide has to be distinguished from other erythematous eruptions.

From the mottling due to cold it differs in that the mottling or "livedo" is a passive congestion and takes the form of a network corresponding to the distribution of the veins in the skin, while the roseola is an active erythema in patches corresponding to the arterial zone, that is, to the meshes of this network.

Toxic erythema due to drugs, such as copaiba and antipyrin, or to food poisons, is distinguished by its more vivid colour, the sharp margins of the patches, their irregularity in size and distribution. They are often grouped together or actually blended into large masses which the roseola never is, or they may be more profuse on the face and limbs. Moreover, a toxic erythema often itches; the syphilide never.

From pityriasis rosea (to be described in the next section) the roseola is easily distinguished by the absence of scaling in the syphilitic eruption.

Other Symptoms which may accompany the Roseola and aid the Diagnosis.

Although we may often see an early case of syphilis without roseola or with that eruption very faintly developed, a syphilitic roseola is seldom present without other signs of syphilis.

Other signs and symptoms to be particularly sought for are: Shotty hardness of the epitrochlear glands and of the glands in the groin and neck; general malaise with headaches, anæmia, joint pains, febrile disturbance, and sore throat; and remains of the primary lesion.

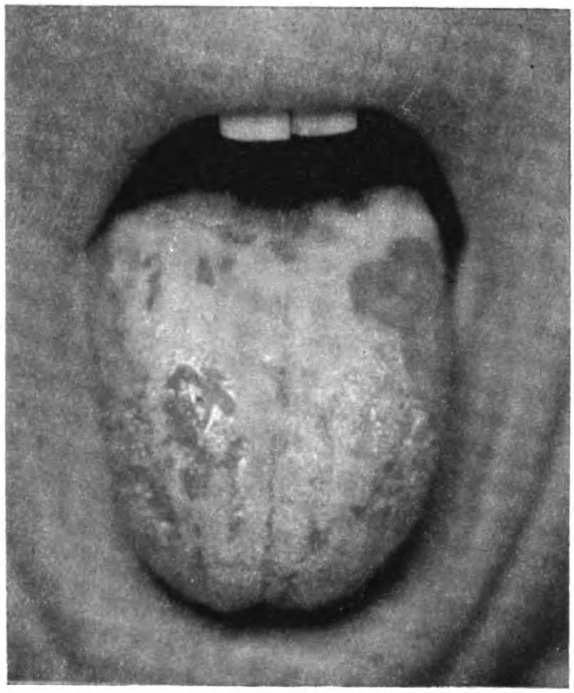

FIG. 2.—"Smooth patches" on the tongue sometimes associated with the macular syphilide.

But there are still other useful confirmatory signs which we may look for, which are due to the same causes which produce the roseolar patches, of which they may be regarded as modifications due to position.

On the *tongue* there may be smooth, red, oval patches which are really macules on a mucous membrane (Fig. 2).

On the *face*, in a small proportion of cases, the roseola may appear about the nose and mouth, and then it presents a slightly raised and scaly margin, constituting the early *circinate syphilide* (Fig. 3).

On the *neck*, in women especially, in whom the skin in this region is generally dark, there may be rounded or oval white areas or patches, known as *leucoderma syphiliticum*. Such patches are on the site of former roseola macules and are produced by the fading of these. They become more noticeable in course of time as the roseola dis-

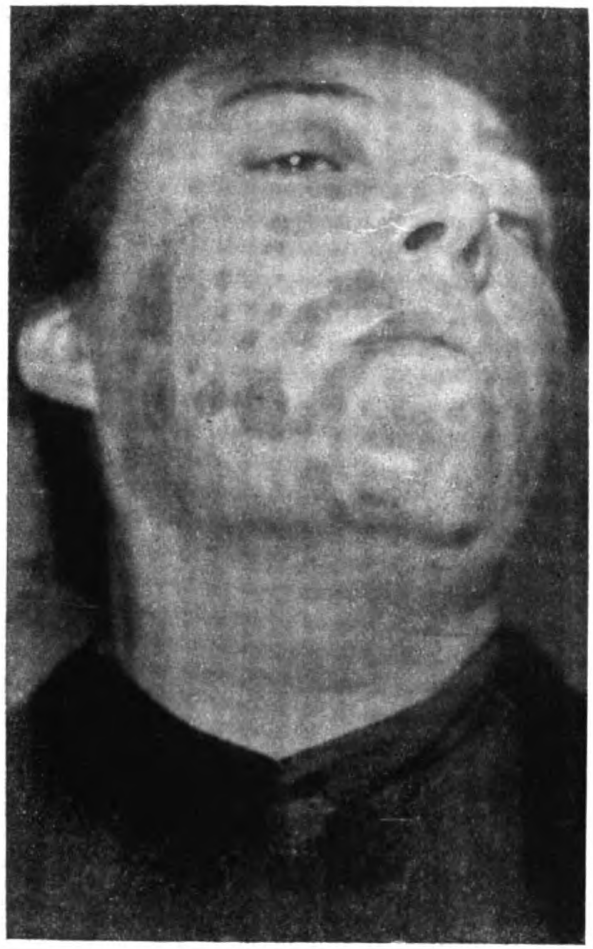

FIG. 3.—Early circinate syphilide on the face associated with the macular syphilide.

appears, and persist for months or years, so that they form a valuable sign of syphilis even after all secondary symptoms are gone (Fig. 4).

On the *trunk*. Careful inspection of the macules on the trunk may show that some of them present a goose-skin appearance from the formation of tiny red papules round the hair follicles included in the macule. This condition is the early stage of a later eruption known as the grouped follicular syphilide or *lichen syphiliticus* (Fig. 5), and its presence is of great help in distinguishing a syphilitic roseola from a toxic erythema.

On the *scalp* there may be a patchy form of baldness distinguished from alopecia areata by the fact that the patches are not so sharply circumscribed as in alopecia areata. The eyebrows and the eyelashes may also be thinned in patches. In this patchy fall of hair the bald areas no doubt correspond with areas which would be roseola macules elsewhere and are due to the toxic action of the spirochætes locally present.

On the *palms* the occasional presence of macules has already been noted as a diagnostic help.

II. The Papular Syphilide.

The other common syphilide of the early months of syphilitic infection is the *papular syphilide*. This appears later than the roseola, from two to four months after infection. Its onset sometimes overlaps the roseola, so that the two eruptions may be present together, their existence affording an important aid to diagnosis (Fig. 6). The eruption

FIG. 4.—White patches (or leucoderma syphiliticum of the neck) which result from the fading of the macular eruption.

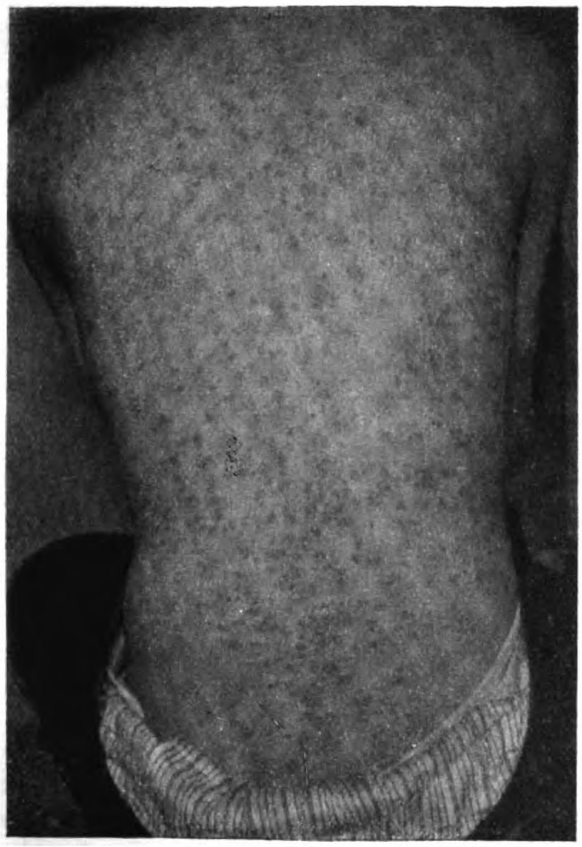

FIG. 5.—Lichen syphiliticus, an eruption of small follicular papules in groups, each group corresponding with the site of a previous macule.

consists of rounded or oval, copper-coloured papules or nodules, which feel distinctly infiltrated on palpation. The coppery tinge and the infiltration are noteworthy characters, which are of help in the recognition of the eruption. It is often widespread and profuse over the trunk, the limbs, and the face. On the face it attacks especially the forehead, the more adjacent parts of the cheeks, and the chin. On the limbs it is generally more profuse on the flexor surfaces. Sometimes it is more sparsely distributed, and then its appearance, particularly in certain favourite positions—the forehead, the face round the mouth and nose, about the genitals, and on the palms and soles—is also a help towards diagnosis (Fig. 7).

But the eruption is not confined to the skin; it generally involves also the mucous membranes, particularly the fauces and tonsils, the tongue, the inner side of the lips and angles of the mouth, the margin of the anus and the inner surface of the vulva, producing in these parts so-called *mucous tubercles* and ulcerations, which are really papules on a moist surface.

All the eruptions of this period are modified forms of this common *papular* or *lenticular syphilide*. The *nummular syphilide* is merely a name given to the eruption when the lesions are very large and flat (Fig. 8). Other types are due to the tendency in some cases to form scales, or crusts, or, less often, to ulcerate. Such scaling or crusting may affect only a few papules or the majority. When most are scaly it is often described as a *psoriasiform syphilide* (not syphilitic psoriasis); when crusted, as an *ecthymatous syphilide*. Sometimes a few or several of the papules or nodules of a secondary syphilide may ulcerate. The presence of such ulcerating syphilides is of great help to diagnosis on account of the characteristic appear-

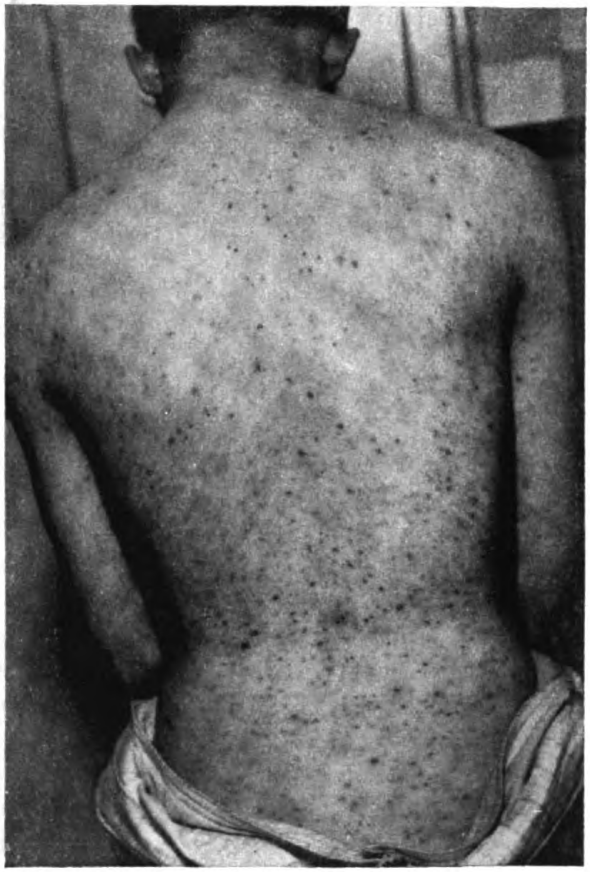

FIG. 6.—Macular syphilide (represented by the paler patches), with commencing papular syphilide (the darker patches).

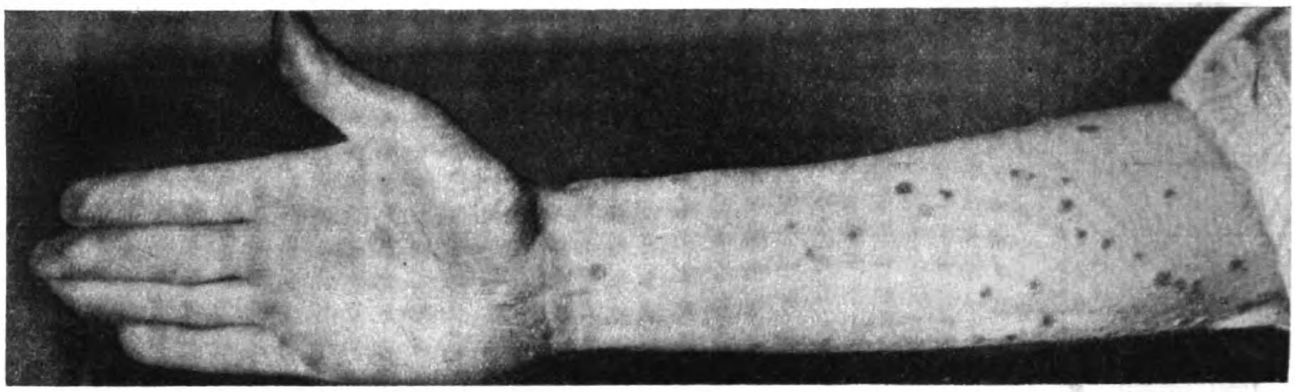

FIG. 7.—Papular syphilide on forearm and palm.

ance which the ulcers present. The central part of the papule ulcerates, but a narrow margin of the papule remains, so that we see an ulcer or crust with a narrow ring-like collar or border (see Fig. 9).

Two special forms of syphilide may occur at this period. They are *small follicular syphilide* (or lichen syphiliticus) and the *corymbose syphilide*. These are papular syphilides, having a special grouping or arrangement of the individual lesions. In the *small follicular syphilide* the papules are pin-head size and seated around the opening of a

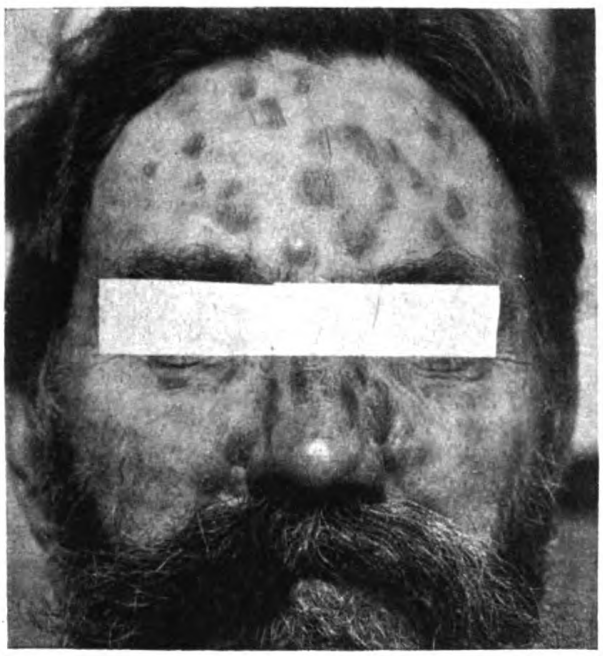

FIG. 8.—Nummular syphilide. Similar papules were present, scattered over the trunk and limbs. This photograph shows a characteristic distribution of secondary syphilide on the forehead and at the sides of the nose.

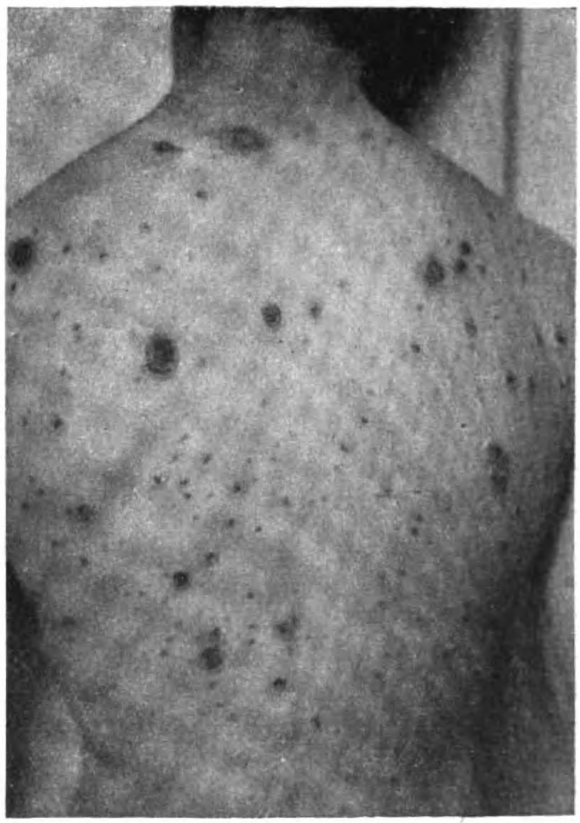

FIG. 9.—Papular syphilide, showing ulceration of some of the lesions with characteristic narrow non-ulcerated border well seen in large lesion over left shoulder-blade.

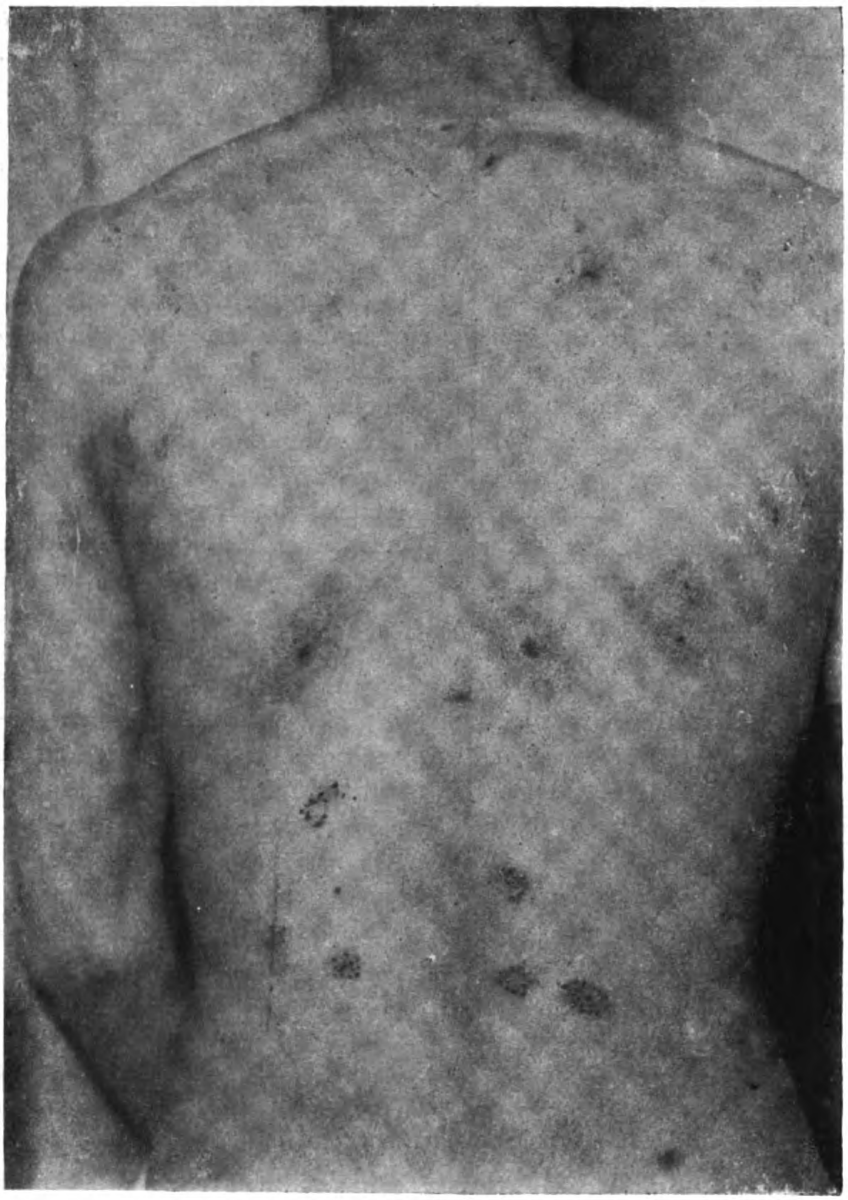

FIG. 10.—Corymbose syphilide, showing central large papule with smaller papules grouped closely around it.

hair follicle. They occur in groups of a score or more, and such groups are scattered more or less thickly over the trunk (see Fig. 5). An interesting feature of this eruption is that it originates from the roseola macule, as has already been explained. It is a very persistent eruption, and may last for many months in spite of treatment.

The *corymbose syphilide* is a papular eruption made up of large and small papules in groups or patches, each group consisting of a central large papule with a profusion of smaller papules around it. It is not very common, but when it occurs it can be mistaken for no other skin eruption (Fig. 10).

(*To be concluded.*)

July 5th, 1915.

CLINICAL JOTTINGS.

(*Continued from p.* 208.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

Forms of Uræmia.

Uræmia is commonly described as occurring in two forms—acute and chronic. In the one the symptoms are of acute onset, of great severity, and of grave prognostic significance. In the other they are of gradual onset, of less apparent severity, and of very indefinite character. These two groups are often called acute and chronic uræmia respectively. They stand in such strong clinical contrast that it would be well if different names were used to designate them. For the acute cases "acute uræmia" might be retained; for the chronic "chronic renal toxæmia" might be a better term.

By chronic uræmia, or chronic renal toxæmia, is meant that gradual failure of health and nutrition, with the various and often indefinite symptoms which are met with, especially in the later stages of granular kidney, but in some degree also in the later stages of chronic parenchymatous nephritis.

Closely allied to this group, and equally distinct from acute toxæmia as ordinarily understood, are the symptoms associated with total or almost complete suppression of urine, such as is met with sometimes in acute nephritis, but more commonly where the excretion of urine is prevented, either by complete obstruction of both ureters, or after one kidney has already been destroyed by disease or surgical operation by obstruction in the other. In these cases the patients die in eight or ten days of rapidly increasing asthenia, but, as a rule, without fits or acute uræmic symptoms.

From both these conditions acute uræmia is clinically distinct. The great characteristic is the suddenness with which the symptoms arise. Without any warning the patient is seized with twitchings; which rapidly develop into violent epileptiform convulsions. The patient becomes unconscious, and dies in a few hours unless the fits cease.

In acute nephritis the symptoms often subside, so that the prognosis is by no means so hopeless as the condition of the patient during the fit might suggest. Indeed, recovery sometimes seems almost to date from the attack.

With granular kidney, on the other hand, acute uræmia is almost invariable fatal.

The Diagnosis of Uræmia.

In acute nephritis, when the patient is already under observation, the diagnosis is easy, for the attack usually takes the form of fits and coma.

In granular kidney the diagnosis is by no means so easy, especially if the attack of uræmia be, as it often is, the first sign of serious disease. Thus it is not uncommon for the patient to be found unconscious and dying, without the diagnosis being certain between uræmia, epilepsy, and apoplexy. Moreover, in granular kidney the symptoms of acute uræmia have by no means the definite character which is often assumed. The cases really vary from one another almost as much as do cases of so-called diabetic coma.

Fits and coma are the two most characteristic symptoms. Yet patients may have no fits, or, at any rate, no marked convulsions, and they need not be comatose. A patient may be comatose without fits, or at most with only slight twitching, the condition closely resembling apoplexy. In other cases the condition closely resembles that of narcotic poisoning. In some instances the most profound collapse may suddenly develop, as if the patient had been suddenly poisoned, and the symptoms are then very much like those met with in ptomaine poisoning.

Frequently signs of cerebral irritation develop, the patient becomes extremely restless, sleepless, more or less delirious, and sometimes passes into a condition of noisy active delirium, simulating delirium tremens, with which it may be confused. At other times the patient becomes for the time violently maniacal. If death does not supervene the attack may subside, and in a day or two pass off. But even then the general condition rapidly deteriorates, and other symptoms develop which before long end in death.

The most interesting fact about acute uræmia in the course of granular kidney is that it so often develops without any warning in the midst of apparent health.

(*To be continued.*)

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE

ON

THE SKIN ERUPTIONS OF SYPHILIS AND THEIR DIAGNOSIS.

By H. G. ADAMSON, M.D., F.R.C.P.,

Physician for Diseases of the Skin at St. Bartholomew's Hospital, London.

(*Concluded from p. 223.*)

II. THE PAPULAR SYPHILIDE (*continued*).

Diagnosis.

In the diagnosis of syphilides in the later secondary stages we have then, as far as the eruption is concerned, to rely upon:

(1) The coppery colour. (2) Infiltration. (3) Distribution on face, trunk, genitals, and flexor surfaces of the limbs—a coppery papule on the palm is of especial value as a diagnostic sign. (4) Mucous membrane lesions. (5) The presence sometimes of a fading roseola, of grouped follicular papules, or of leucoderma syphiliticum. (6) The character (described above) of the ulceration, if present. As other evidence of syphilis we may get general malaise, anæmia, general gland enlargement, remains of the primary sore, patchy alopecia, iritis, deafness, albuminuria.

There are three eruptions which one ought to bear in mind as most commonly mistaken for those of secondary syphilis. These are: Pityriasis rosea, psoriasis, and lichen planus.

Pityriasis rosea is a common eruption with characteristic features, but not very well known among medical practitioners and often mistaken by the unwary for an early eruption of syphilis. The real nature and cause of pityriasis rosea are not known. It attacks healthy people of all classes.

The first sign is a pinkish, scaly patch on the trunk which suggests a ringworm. After a week to ten days there is a sudden eruption of similar patches over the trunk and upper parts of the arms and thighs. The eruption generally lasts for several weeks and then disappears. Its characteristic features are: (1) Its sudden onset following the "herald patch"; (2) its "vest-area" distribution; (3) the peculiar type of scaliness of the patches, due to the fact that desquamation begins at the centre of the patch and spreads out to the margin, so that eventually there is a central, wrinkled, faded yellow area with a pinkish desquamating margin; (4) itching, which is more or less intense.

Psoriasis is another eruption which is sometimes mistaken for the papular eruption of secondary syphilis. It is distinguished by the more silvery character of the scales and by the entire absence of infiltration after the scales have been scraped off, which is in contrast with a syphilitic papule, which always shows infiltration, even after scraping. The good general health of a psoriatic patient is in contrast with the anæmia and general malaise of a syphilitic. The history of previous similar attacks of psoriasis or of psoriasis in other members of the family is also of great value in making a diagnosis. Sometimes, psoriasis being a very common eruption, we may see a syphilitic eruption and a psoriasis on the same patient. With a little care it is generally not difficult to distinguish the patches of psoriasis from the papules of syphilis, for the eruptions retain their distinctive characters, and neither eruption is modified by the presence of the other disease.

Lichen planus is sometimes mistaken for syphilis, but the characteristic distribution about the wrists and inner sides of the knees and legs, the elementary lesion—the flat-topped, shiny, angular, lilac-coloured papule—and the presence of intense itching should distinguish that disease.

III. Eruptions of the Intermediate Stage.

During the years between the period of active syphilis, the so-called secondary period, and that of late or tertiary syphilis, the patient may be troubled with certain eruptions which have been termed "reminders." Such are mucous patches or ulcers upon the sides of the tongue or on the fauces; scaly affections of the palms; scaly papular eruptions about the genitals; affections of the nails; and papular eruptions on the face, trunk, or limbs. The most characteristic feature of these eruptions are that they tend to be asymmetrical or localised to certain areas, particularly the face, the genitals, the palms, and the mucous membranes; or that they take a circinate or horse-shoe shape—hence the name "*orbicular syphilide*" (Figs. 11, 12, 13).

It is hardly possible to mistake these eruptions for any other. That the rings or broken circles are made up of a chain of separate flat nodules with coppery colour and tendency to dry scaling (or mild ulceration in moist parts) is sufficient to distinguish them from psoriasis, from lupus, or from ringworm, with which diseases they might be confounded by the casual observer.

Another feature of these "reminders" is that they are apt to recur in the same locality after removal by treatment.

Fig. 11.—Recurrent "orbicular syphilide" of the period between the secondary and tertiary eruptions.

FIG. 12.—Orbicular syphilide of the palm.

IV. ERUPTIONS OF LATE OR TERTIARY PERIOD OF SYPHILIS.

The skin eruptions of the later period of syphilis are more uniform in character, and, therefore, generally more easy to recognise than the earlier syphilides. They are of two types, the grouped nodular or *papular tertiary syphilide*, and the *gummatous syphilide*. These two types are really different degrees of the same eruption, and intermediate stages link them together. All tertiary syphilides have certain characters in common; they are circumscribed inflammatory cell infiltrations, which tend to break down and produce punched-out ulcers and scars; *the nodules or papules and gummata tend to occur in groups or broken circles.*

The common *papular* or *nodular tertiary syphilide* is usually easy to recognise on account of the tendency of the nodules to be grouped into patches or circles while remaining separate from one another (Fig. 14).

The nodules are of a dull coppery red colour. They may be smooth, or scaly, or crusted. Generally, some in the group are ulcerated, the ulcers being crusted or not; and sometimes scars are present as well. This combination of papules, ulcers, and scars in the same group is characteristic. The scars left by the healing of lesions have, too, the same grouping of distinct and separate scars, which often enables one to recognise them immediately as of syphilitic origin (Fig. 15).

The groups of nodules are usually single or few and distributed without symmetry; occasionally there may be *many* groups scattered over the

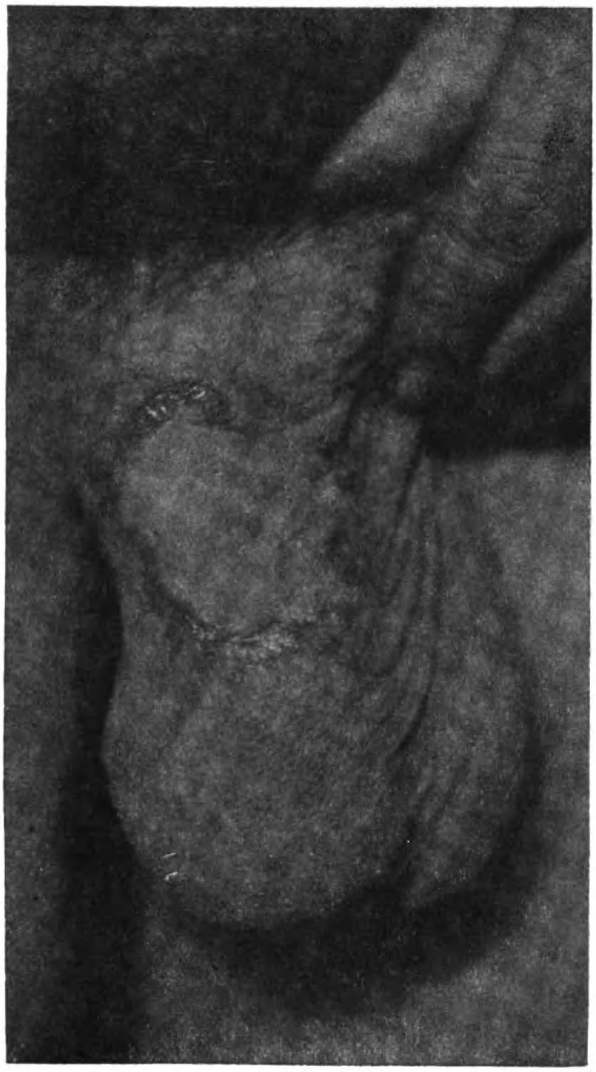

FIG. 13.—Ulcerating orbicular syphilide of the scrotum.

body and limbs—a circumstance which may put one off one's guard and lead to a wrong diagnosis. Often certain favourite sites are attacked, particularly the nose and central parts of the face (Fig. 15), the genitals and palms (Fig. 16)—or rather palm, for tertiary syphilis here, as elsewhere, is usually asymmetrical.

It is important to remember that there may be no obtainable history of syphilis previous to the tertiary eruption, and this is especially so in women, in whom the early symptoms may have been really absent. But there may be history of miscarriages in women, and there may be chronic superficial glossitis in men, as aids to diagnosis.

Diagnosis.

The eruption for which a tertiary syphilide is most often mistaken is that of lupus vulgaris—often because there is no history of syphilis obtainable, and the subject is thought an unlikely one to have had syphilis. The onset of lupus, generally in early life, and its slow progress on the one hand; the onset in adult life and the comparatively rapid spread on the other, help to distinguish lupus from tertiary syphilis. But the most important difference is in the lesions themselves. The nodules of lupus are small, fused together, and leave "apple-jelly" infiltration on glass pressure. Those of syphilis are large, separate, though grouped; each

FIG. 14.—Tertiary papular syphilide, showing characteristic grouping of the nodules.

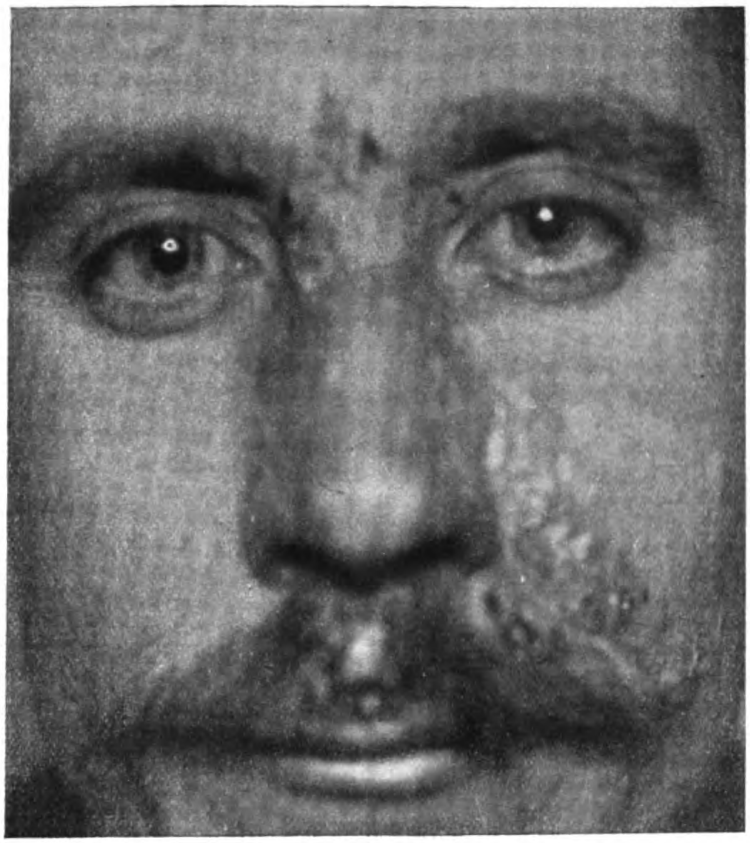

FIG. 15.—Grouped scars about the nose and central parts of the face following a tertiary syphilide.

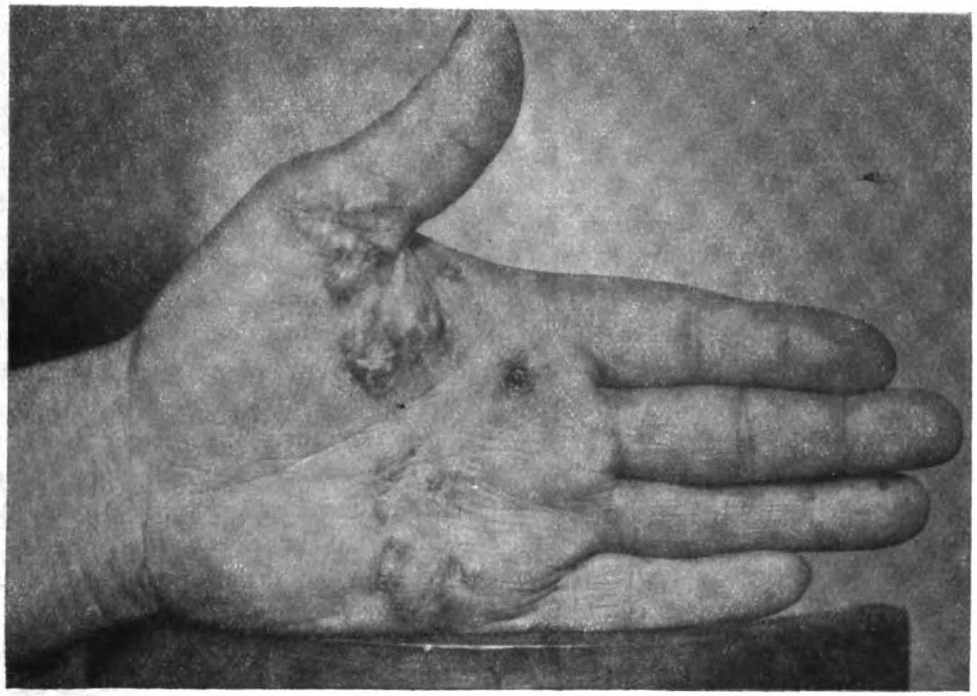

FIG. 16.—Ulcerating tertiary syphilide of the palm—here again the ulcers are characteristically grouped into broken circles.

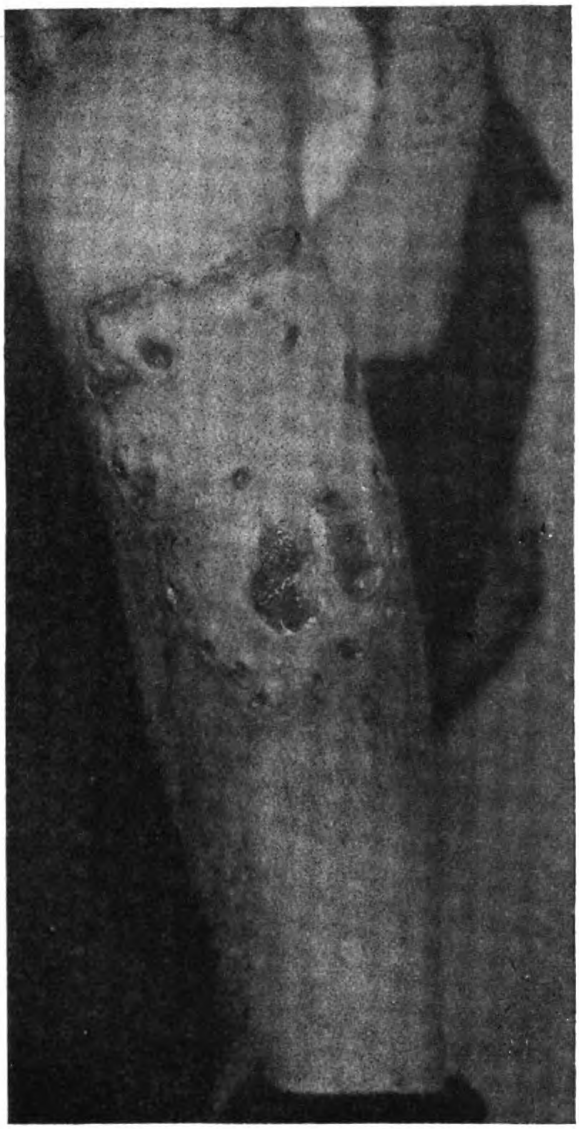

FIG. 17.—Tertiary syphilide—grouped ulcers, each ulcer the result of the breaking down of a papule or nodule.

nodule ulcerates separately and leaves a separate scar, and they do not present the characteristic apple-jelly nodule on firm pressure.

Chronic psoriasis patches are sometimes very difficult to diagnose from tertiary syphilis, because they sometimes take a circinate grouping, and because the nodules of a tertiary syphilide may be superficial and scaly. The presence of some infiltration, and a tendency, however slight, to ulceration and scarring is to be looked for as indicative of syphilis.

Gummatous syphilides are really large nodules which soften and break down to form punched-out ulcers (Fig. 17). The eruptions most often mistaken for them are ulcers following a streptococcal skin infection, and certain tuberculous ulcers which occurred on the legs in young women, and are known as "Bazin's disease." Again, the most important point of distinction is that the gummatous ulcers always show some sign of being made up of several separate gummata, which are grouped together or blended in a horse-shoe curve or broken circle.

Although the diagnosis of tertiary syphilis is, as I have said, generally comparatively easy, we may sometimes meet with cases where we are in doubt whether we have to do with syphilis or with tuberculosis. For these cases we may get valuable help from the results of a Wassermann test and a tuberculin "cuti-reaction."

But these tests must never take the place of clinical observation and experience, for we must remember, because a patient gives a positive Wassermann or positive tuberculin reaction, it does not always follow that the eruption we are dealing with is syphilitic in the first instance or tuberculous in the other.

The results of treatment with anti-syphilitic remedies is, perhaps, an even more practical test in a doubtful case than the Wassermann or tuberculin reaction tests.

July 5th, 1915.

Ligation Method of Treating Varices.—Secher has followed the technique introduced by Schede and Kuzmik, and extols it as simple and free from danger, while it takes comparatively little time and the result is excellent. The operation is easy, thus is particularly useful for the general practitioner. Under general anæsthesia a silk suture thread or catgut is passed around under the vein, without exposing it, and this is repeated at intervals along the vein. The ends of each thread are then tied over a roll of gauze or rubber tube, and the patient stays in bed one or two weeks. Schede applies the catgut threads at 2 cm. intervals, and removes them after two or three days, and lets the patient get up with a bandage the eighth day. Kuzmik uses silk at 5 cm. intervals, and leaves the threads for twelve days.—*Journ. A.M.A.*, lxiv, p. 1803.

CLINICAL JOTTINGS.

(*Continued from p.* 224.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

Rheumatic Fever and Rashes.

Various rashes are described as occurring in the course of rheumatic fever. Yet there is only one which is common, viz. the sweat-rash or sudamina; all the others are rare. They are for the most part different forms of erythema. The frequency with which these rashes are stated to occur in rheumatic fever is very variously given by different writers. This depends upon the degree of laxity with which the term "rheumatic fever" has been used in the past. Rheumatic fever is no doubt a specific disease with its own microbe, though the actual specific organism may perhaps not yet have been discovered. The mere association of raised temperature with joint pains is of course not sufficient to justify a diagnosis of rheumatic fever, for it is met with in so many forms of septic infection of other kinds. Gonorrhœa may be taken as an apt illustration. In the early days of tuberculin treatment joint pains and swelling commonly followed the injections, and they might with equal propriety have been called rheumatic, but they never were. The same difference, and for the same reason, used to be seen even in the statistics of *morbus cordis* as a complication of rheumatic fever, which was stated by some writers to be only about 3 per cent. and by others as much as 70 or 80 per cent. If the diagnosis of rheumatic fever be more definite and more carefully and correctly made, as it is now, the liability to *morbus cordis* is found to be very greatly raised even to as much as 50 per cent. in the first attack of the fever, while the frequency of the skin eruptions is reduced very low, almost, except for sudamina, to a vanishing point. *Per contra* the erythemata are not infrequently associated with joint and muscle-pains and rise of temperature, but they rarely result in any cardiac affection. This is especially true of erythema nodosum, which is still often called rheumatic because of the pains and fever; yet I have never seen erythema nodosum occur in the course of rheumatic fever, nor have I myself observed *morbus cordis* develop in the course of erythema nodosum, though I know such cases have been described.

Most of the rashes which have been described as occurring in rheumatic fever are of the erythema type and are of little or no importance. One only need be further considered, and that is the hæmorrhagic or purpuric form. All the erythemata are attended with some extravasation of blood-colouring matter into the skin, and therefore lead to staining, which persists for a time after the rash has gone, and now and then the extravasation is sufficient to justify the term "hæmorrhagic." I have never seen such a hæmorrhagic erythema in the course of rheumatic fever, though it is often associated with joint pains and raised temperature, as is suggested by the name, peliosis rheumatica, which is given to it.

Peliosis rheumatica is not rheumatic fever at all, but a grave and often fatal septic infection of an entirely different nature. If associated with any affection of the heart the lesions are of the ulcerative, malignant, endocarditis type. In fact, peliosis rheumatica might often be well described as malignant septic purpura as distinguished from purpura simplex. It is, at any rate, entirely distinct from rheumatic fever.

The Significance of Albuminuria.

Albuminuria is a symptom, and not a disease.

Its significance depends upon the disease of which it is a symptom.

In treatment it is the disease or cause which is to be treated, and not the albuminuria. Even if the albumen could be made by treatment to disappear from the urine while the cause that produced it persisted, the patient would not necessarily be better. But this can rarely be done, and the albuminuria continues till the disease upon which it depends is treated, and then it may diminish or disappear.

To attempt by diet to starve, as it were, the albumen out, would starve the patient too, who might easily suffer more from the want of food than from the loss of albumen. The actual amount of albumen lost in the twenty-four hours is at the highest not more than an ounce or two, and usually it is not many grains. The body is not likely to feel such a loss much, and if the health suffers it is not the mere loss of albumen which explains it.

PHYSIOLOGICAL PRINCIPLES IN TREATMENT. By W. LANGDON BROWN, M.A., M.D.Cantab., F.R.C.P., Assistant Physician to St. Bartholomew's Hospital, and Physician to the Metropolitan Hospital. Third edition. London, 1914. Baillière, Tindall & Cox. Pp. 408. Price 5*s.* net.

The author describes the applications of recent physiology in the treatment of disease in a clear and most interesting fashion and shows that there is a closer harmony between physiology and practical medicine than is generally supposed. As examples may be taken the applications of Gaskell's work on the heart by Mackenzie and others, the introduction of convenient methods of registering blood-pressure in man, the increased knowledge of the chemistry of uric acid and its congeners, nitrogenous metabolism and internal secretion. The busy practitioner who has forgotten much of his physiology and has not had time to keep abreast of these advances will find them here presented concisely and lucidly. His knowledge of therapeutics will be corrected, and he will be saved from many hoary fallacies, and the bad and meddlesome practice dependent on them. Thus the author proves that if any protein food is absorbed from ordinary nutrient enemata, the amount is so little as to make it hardly worth while to subject patients to the discomforts of the method. In the treatment of nephritis he shows that the idea of limiting the protein in the diet in order to diminish the albuminuria is bad, because it cannot effect this object and deprives the patient of essential nourishment. The fallacy of urea estimations as a guide to the renal capacity is thus exposed: "The procedure often adopted is irrational. A man with chronic nephritis on a restricted diet has his urea estimated. Instead of the normal 30 grm., he is found to be passing, say, only 16 grm. The physician concludes that the capacity for urea excretion must be seriously decreased; the patient must take less nitrogenous food. This is done, and the next analysis shows an even lower urea excretion. 'Worse and worse,' thinks the physician; 'this man is only fit for a milk diet.' Accordingly he is given three pints of milk a day and nothing else. He now bids fair to fulfil the gloomy prognosis formed, unless he fortunately rebels against this pitiful fare and takes the law into his own hands. For, as the greater part of the urea comes direct from the food, the more the nitrogenous food is restricted the less urea will be excreted."

This edition has been thoroughly revised throughout. The principal new points are as follows: In the chapter on organotherapy the pituitary section is rewritten so as to give the main points in Cushing's important work. A new chapter on the mechanical factors in digestion and indigestion is inserted, in which the question of visceroptosis is discussed. The new tests for pancreatic inadequacy are described in the chapter on the work of the pancreas. The chapter on glycosuria and diabetes is rewritten. The chief points learned from the electro-cardiograph are briefly discussed and some tracings given. The influence of vitamines in disease are considered.

It will be seen that this book should be in the hands of every practitioner who desires to give his patients the advantages of up-to-date therapeutics.

SURGICAL MATERIALS AND THEIR USES. By ALEX MACLENNAN, M.B., C.M.Glas. London: Edward Arnold. Pp. 250. Price 4*s.* 6*d.* net. 1915.

This book is divided up into six sections, viz. bandaging, splints, dressings, antiseptics, sutures and ligatures, and instruments. There are over 270 illustrations which allow the text to be followed with the utmost ease. Under each of these headings will be found quite a mine of information for the medical tyro, as well as one from which the experienced practitioner can draw with both pleasure and profit. Where all is so good it is difficult to particularise. Nowhere, perhaps, is the march of surgical progress more defined than in regard to dressings, sutures and ligatures. The morbid conditions which require wet dressing as compared with dry are detailed and will repay close study. On the vexed question of the use of gloves in operating the author states that: "As a protection against infection the use of gloves may give rise to a factitious sense of security and should not altogether be relied on." On the only occasion that he was infected during an operation he was wearing gloves. He introduced their use at the earliest opportunity in 1898 and, with the occasional use of cotton gloves, has used rubber gloves ever since. An excellent index concludes a book for which we have nothing but praise.

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We regret that owing to the war the number of pages must be diminished for a time.

CHRONIC GASTRIC ULCER.

By ALBERT J. WALTON, M.S., F.R.C.S.,

Assistant Surgeon to the London Hospital.

OF late years, owing to the fact that cases of chronic gastric ulcer are more frequently treated surgically, the diagnosis is better controlled, and hence the symptomatology is more accurately defined. Errors of diagnosis do, however, still occur.

During the last two years I have personally operated upon 73 cases of chronic gastric ulcer. Of these 18 were admitted for acute perforation, while the remaining 55 presented chronic symptoms. It is this latter group which I propose to consider at the present time, and to analyse the symptoms which they presented.

PATHOLOGY.

A typical chronic gastric ulcer is usually represented even in modern text-books as being irregular in outline and occupying a relatively large area of the stomach. This is the condition not infrequently found post mortem. In my experience, however, such an ulcer is rarely seen at an operation. Of the 55 cases 44 presented an ulcer circular in outline, and varying from a quarter to one inch in diameter, the majority not being more than half an inch across. These ulcers were deeply punched out, and extended in every case through the muscular coat, so that on microscopic examination there was no evidence of remaining muscle tissue. The area around was hard and indurated, so that a portion of the stomach, one to one and a half inches in diameter, could be picked up between the thumb and finger as a hard mass distinct from the normal stomach wall. In some such cases the peritoneum was bare, but had the characteristic stippled appearance. If

situated near the lesser curve there was often a fatty mass of adherent omentum, whereas if on the posterior wall they were not infrequently attached to the pancreas. In every case a finger invaginated through the anterior stomach wall could distinctly feel the well-marked depression of the ulcer. It is of interest to note that the majority of these ulcers were situated at or about the lesser curvature, there being 30 in this position, and only 14 at the pylorus. In the second group of 11 cases the ulcer was larger, more irregular, and not so well defined from the surrounding stomach area, thus conforming to the conditions usually described. It might be thought that this difference was due to the fact that the surgical ulcers are seen at an earlier date than those described at post-mortem examinations. It is doubtful, however, whether such is the case, for it not infrequently happens that a patient with a well-defined small ulcer has a clinical history extending over 10 or 15 years. Of the large irregular ulcers a higher proportion was situated at the pylorus than was the case with the small ulcers, 5 of the 11 being found in this position. Of the total of 55 cases, therefore, 36 ulcers were situated at or about the lesser curvature at a distance varying from 2 to 4 inches from the pylorus, and 19 of them were placed in close relationship to the pylorus.

In this small series gastric ulcer was found much more commonly than duodenal ulcer, for, in the period of time corresponding to that in which the 73 gastric ulcers were operated upon, only 30 duodenal ulcers were seen. As is usual, the condition was found to be more common in males than in females, 50 of the 73 cases being in males.

Symptoms.

The majority of cases showed characteristic symptoms of definite attacks extending over a prolonged period, the patient being perfectly free in the intervals. These attacks had occurred over periods extending from 2 to 40 years, the attacks themselves lasting 1 to 2 weeks, and the intervals varying from 2 weeks to 4 or 5 months. It occasionally happened, however, that the attack would last for 3 months, and not uncommonly it was found that the last one, namely, that for which the patient sought advice, had persisted for a longer period than the previous attacks; so much so that it occasionally gave rise to the suspicion of carcinoma when only a simple ulcer was found at operation. It was also found that in some of these cases the symptoms during the last attacks had become much more continuous, and that the appetite had even been decreased, so that the clinical picture of carcinoma was very closely simulated. The age of the patient was of but little help in differentiating between the two conditions, for, although the average was 42, 2 patients were over 70, 1 between 60 and 70, and 9 between 50 and 60. It is true that in some of these cases the symptoms had existed for 20 or 30 years, yet in others they were of much shorter duration, and in the case of the 2 patients over 70 the symptoms had been present for 2 and 15 years respectively, so that in both cases they had commenced well past the age at which carcinoma is commonly seen. Moreover, even with a long history, such symptoms might be due to the onset of carcinomatous changes in a simple ulcer of long duration.

In the same way that cases were occasionally seen with attacks which lasted longer than usual, so also it would happen that in some the interval between the attacks would be considerably prolonged. Thus, in one instance, a patient aged 58 years, the first attack had occurred 33 years ago, and the patient had then remained perfectly free for 32 years, after which an attack had occurred which lasted for 12 months before he sought treatment. At operation a characteristic ulcer about half an inch in diameter was found on the lesser curvature. Excision of the ulcer, combined with the posterior gastro-enterostomy, has been followed by complete relief from all symptoms. In 4 cases there have been intervals of freedom of over 2 years. The fact that the intervals between the attacks may occasionally extend over a prolonged period, and that in this interval the freedom is complete, and the fact already pointed out that in so large a proportion of this series the ulcers were small and showed certain acute characteristics, is very suggestive that the ulcers heal entirely during the intervals, and that it is the tendency for ulcer formation rather than the ulcer itself which persists for so long a period. The condition in which the ulcer progresses month by month and year by year,

until a large portion of the stomach is affected, is, in my experience, extremely rare.

The individual symptoms are very distinctive, and it is but seldom that a chronic gastric ulcer will be diagnosed as any other lesion. As already mentioned it may occasionally simulate carcinoma, but even here the symptoms are generally such as to lead to a suspicion of carcinoma rather than to the actual diagnosis of this latter condition. Since both conditions will require operative treatment but little harm is done. There is, however, a greater tendency to make the diagnosis of gastric ulcer when no such lesion is present. A condition which I think can be best described as "visceroptosis," and which is variously known as "intestinal stasis," "chronic abdomen," or "intestinal kinks," generally presents, as I shall hope to show in a future communication, one of three groups of symptoms. One of these groups, namely, that frequently occurring in adult women, very closely resembles gastric ulcer, and the difficulty is greatly increased by the fact that not uncommonly the two conditions are found in association, so that although there is sufficient for one to be able to definitely say that visceroptosis is present it is at times difficult or impossible to be certain that there is not a concomitant gastric ulcer.

The symptoms which are found in the individual attacks may be considered as follows.

Pain.—This is usually described as being severe, and appears from a half to three hours after food. In a previous communication * I have discussed the evidence brought forward to show that the time of onset of the pain is dependent upon the position of the ulcer. This present series of cases does not, however, support this contention, for in 14 cases the pain appeared late after the meal, not commencing until two hours after the meal was completed, but of these 14 cases 8 had the ulcer on the lesser curvature, although in two of them it was within one and a half inches of the pylorus. Six only of them had a definite pyloric ulcer. In all cases the pain was a very definite factor. The majority gave a history of mild pain in the early stages, but it was in all severe when the patient sought treatment. Although situated in the epigastrium it tended to radiate, and this was especially so where the symptoms had existed for many years. Thus of the total number of cases, 55 in all, 24 showed pain radiating to the back and left shoulder, but in those which had persisted for ten years or more, 32 in all, 23 showed radiating pain, or, in other words, of the 24 cases with radiating pain 23 had symptoms lasting over ten years. The pain is often of great severity, so that the patient may be doubled up, and at first sight the condition may resemble colic. This is more likely to be so if the ulcer is adherent to the pancreas, and here the pain is not only more continuous during the course of the day, but the attack tends to be longer, the pain at the same time radiating more definitely to the left shoulder, so that it is at times possible, owing to the constancy, the severity, and the radiation of the pain, to determine that the ulcer is adherent to the pancreas before operation.

Another very interesting question is that of the relief of the pain following the ingestion of food. It is a well-known fact that this is a marked symptom in cases of duodenal ulcer. It is, however, occasionally very clearly defined when the ulcer is in the stomach. In this present series it was found to occur in 15 cases. Of these 15 the ulcer was situated on the lesser curvature in 8, but in one of them there was a condition of "hour-glass" stomach which is prone to give rise to anomalous symptoms. In 3 others the ulcer was adherent to the pancreas, a condition which again may alter the normal course of events. There were thus 4 cases of simple ulcer situated at the lesser curvature in which the pain was relieved by the food and 7 cases in which similar relief was found with an ulcer at the pylorus. Hence this symptom is in itself evidence of the ulcer being situated close to the pylorus.

Vomiting.—Of late years more and more stress has been laid upon the fact that vomiting may be absent when a definite ulcer is present. In this series, however, 44 patients had vomited relatively frequently. When there was no pyloric obstruction the vomiting tended to occur at the height of the pain—that is to say, from two to three hours after the ingestion of food—and was more likely to be present if the pain was severe, so that often a history would be given that this symptom had been absent in the earlier stages of the disease. Thus in 4 patients in whom symptoms had been present for from 5 to 10 years vomiting had

* "The Differential Diagnosis of Surgical Dyspepsia," 'Brit. Med. Journ.,' October 5th, 1912.

occurred only in the last one or two attacks, the vomited material consisting usually of the ingested food but little altered in character. Occasionally it would be stated that the vomit was definitely acid in character, and sometimes acid material was alone present without any altered food. The most constant factor of the vomiting is that it immediately relieves the pain, and it was found that in only one out of the 44 cases was this relief not afforded. This one case was complicated by the presence of a second ulcer which extended from the pylorus on to the duodenum, the first ulcer being situated on the lesser curvature.

If the ulcer had led to pyloric obstruction or the formation of an "hour-glass" constriction then the vomited material would be larger in quantity, often ejected forcibly, and perhaps contain portions of undigested food taken one or two days previously. Such vomited material would also frequently show changes due to fermentation.

Hæmatemesis and melæna.—As in the case of vomiting, this symptom, which was at one time regarded as one of the most important pieces of evidence of the presence of an ulcer, is now realised to be frequently absent. In this series of cases it occurred in 26, that is to say 50 per cent. of the total number. In 4 of the 26, however, it was described as being very slight in quantity, and in 3 others it occurred but once at periods of 7, 8, and 10 years respectively before the patient had sought advice. The quantity of blood will vary very considerably. In one man it was so large that immediate operation was carried out mainly for the purpose of checking the hæmorrhage. Generally the hæmorrhage was relatively severe, the patient often stating that he had brought up a half to one pint of blood. In such cases, however, it would tend to cease spontaneously after the first vomit and it was rarely sufficient to give rise to marked anæmia. In fact it not infrequently happened that the most anæmic patients were those in whom there had been no visible loss of blood, the probability being that in these cases there had been a long continued loss of small quantities of blood which had passed unrecognised in the fæces. It is of interest to note here that only one case with an ulcer required treatment owing to the severity of the hæmatemesis, for during the same period of time 6 cases had been transferred to me for urgent surgical treatment owing to the severity of hæmatemesis. In all these no chronic ulcer was seen, and 5 of them were young women. This experience would seem to show that chronic ulceration of the stomach is not a relatively common cause of severe hæmatemesis.

Appetite.—As a general rule, the appetite is unaltered. The patient is definite that his appetite is good, and that he could eat anything, but is afraid to do so on account of the pain. Occasionally, however, it happens that the appetite is poor, and under such circumstances the case is likely to be confused with carcinoma of the stomach. In the present series of cases there were only 4 in which there was a distinct history of loss of appetite. Of these 4, 3 had a distinct decrease of free hydrochloric acid and a decreased total acidity in their test-meals. The fourth case, on the other hand, had an increased acidity in the test-meal. This one, and 2 of the remaining 3, had a condition which, at operation, was diagnosed as carcinoma of the stomach. A partial gastrectomy was, therefore, performed, but microscopic section showed no evidence of growth. In these cases not only was the appetite decreased and the acidity low, both of which conditions would suggest carcinoma rather than chronic gastric ulcer, but the macroscopic appearances strongly supported this view. The fourth case, which did not resemble a carcinoma at operation, had, in addition to an ulcer on the lesser curve, a second ulcer leading to pyloric obstruction, and hence both the decreased acidity and the loss of appetite may be regarded as dependent upon chronic gastritis secondary to the dilatation of the stomach. In 5 cases, which are not included in the above list, there was a good appetite and a high acidity, although an early carcinomatous condition was found grafted on a chronic gastric ulcer.

Test-meal.—Of the 55 cases, 36 had a test-meal administered, and it was found that in 33 both the total acidity and the free hydrochloric acid was slightly or considerably above the normal. In only 3 cases was there a definite decrease in acidity. There has been very considerable discussion as to the value of a test-meal in arriving at the diagnosis. It has been my own experience, as shown by the above figures, that, taken into consideration with the other facts, this test is one of the most valuable at our command. Most of the other conditions which may simulate chronic gastric

ulcer, such as carcinoma of the stomach, gall-stones, or visceroptosis, are generally associated with a decrease in the free hydrochloric acid and in the total acidity, whereas with a gastric ulcer some 91 per cent. have a raised acidity. It has already been mentioned that of the three cases in which there was a diminution, one had a pyloric obstruction, and the other two had a condition resembling at operation carcinoma, there being a very considerable area of induration around the ulcer. It will be noted also that the appetite appears to go hand in hand with the condition of the test-meal. If the test-meal is raised the appetite is good, if it is decreased the appetite is poor, and hence one has a ready explanation as to why the appetite is good in cases of duodenal and gastric ulcers and poor in cases of carcinoma.

Wasting.—Unless there be definite obstruction to the passage of food, as may be seen in pyloric stenosis or "hour-glass" constriction, wasting will not, as a rule, be marked. For, since the appetite is good and the hydrochloric acid tends to be raised, therefore not only will the normal amount of food be taken, but the process of digestion will be fully carried out. In those cases, however, in which there is a diminution in the amount of hydrochloric acid, it has been shown that the appetite is decreased, and it is just in such cases as these that wasting may be present. Of the three cases in this series which have already been mentioned as having a low total acidity, it is noted in all that they were considerably wasted, but it was also found that there were two others which had good appetites and a high or moderate test-meal, and yet were, at operation, suggestive of carcinoma. In both these cases there had been very considerable wasting. In addition to the above, it will occasionally happen that a patient, owing to the severity of the pain, will have been carefully dieting himself for a prolonged period. In some of these the diet may have been so diminished that it was insufficient to adequately support the patient's needs, and in these circumstances wasting may ensue. Here, however, it is usually found that the history of wasting will extend over a period which is often so prolonged that the suspicion of carcinoma will not thereby be raised. Thus, of two patients, one gave a history of marked wasting extending over 5 years and the other over 11 years. In these circumstances the immediate relief which follows operative treatment is associated with a rapid and marked gain in the weight. In one such patient (F. H—) the weight at operation was 9 stone. Eight months later his weight was 13 stone, and a second incision which had been made over the appendix showed a distinct bulge, owing to the rapid increase of his abdominal contents. The gastric incision, however, remained firm. Another patient, not included in this series, who was operated on for a perforated gastric ulcer, gained over 2 stone in weight in less than a year.

Diagnosis.

Visceroptosis.—There is a condition which has given rise to much discussion and which has been described under many names. It is a very definite clinical entity, but I prefer to describe it as visceroptosis. I am strongly of the opinion that the underlying factor is often a downward displacement to a various extent of all the abdominal viscera, a displacement which in many cases is manifestly congenital, while in others it appears to be acquired. At operation it is characterised by a long U-shaped stomach, by a freely movable cæcum and ascending colon, so that these viscera can be readily drawn through the wound, and by the presence of mobile kidneys. Accessory membranes are often found. In nearly all cases there is a definite band on the under surface of the mesentery of the lower few inches of the ileum, the so-called Lane's kink; a well-marked membrane on the upper surface of the cæcum, the so-called Jackson's membrane, and bands of adhesions between the terminal portion of the transverse and the commencement of the descending colon, a condition known as Payr's disease. These membranes, in my opinion, seldom give rise to any mechanical obstruction, but cases with these pathological lesions present very definite clinical symptoms, the cause of which, in spite of the vast amount of literature which has been devoted to the subject, I believe we are still far from understanding. The symptoms appear in several definite groups, of which one alone need be considered here. The patients presenting this group are usually women, about the age of 25 to 40. The symptoms somewhat closely resemble those described above which are found

with chronic gastric ulcer. Often, however, the mimicry is not complete, the intervals of freedom are not nearly so well defined, and not infrequently between the attacks the patient may continue to have vomiting and pain. The pain has no definite relationship to food; it comes on more rapidly after the food and is often not completely relieved before the next meal. The amount of vomiting is more marked than is customary with cases of gastric ulcer, and the pain is often more diffuse. On examination there is definite gurgling in the region of the cæcum, and this distended viscus may be felt. The right kidney may be felt slightly movable. The test meal, however, is frequently of great value, the majority of cases showing absent free hydrochloric acid and a marked diminution of the total acidity. Under such circumstances the differentiation is clear, and no difficulty should arise in determining which lesion is present. Unfortunately, however, such cases may be complicated by the presence of a gastric ulcer, and it therefore becomes extremely difficult, and sometimes almost impossible, to decide when no ulcer is present. Frequently, if this condition of visceroptosis be alone found, the operation is followed by but little improvement. In some few cases the symptoms cease entirely after the division of a band or the removal of an adherent appendix. In the majority, however, there appears to be not the slightest improvement after any operative measures, and I have seen cases which have had the greater part of their large intestine removed with no relief whatever. The condition appears to be very frequent among women to-day, and is a constant source of difficulty in the diagnosis of gastric ulcer.

Carcinoma of the stomach.—A well-defined case of carcinoma of the stomach can easily be differentiated from gastric ulcer. There is a short history commencing in a patient over the age of 35. There is little or no tendency for the symptoms to appear in definite attacks, but rather to be steadily progressive. There is constant, but not severe pain. There is marked loss of appetite and a very distinct difference in the test-meal. In certain cases, however, as already mentioned, patients with gastric ulcer may have a poor appetite, more constant pain, definite wasting, and a diminution in the free hydrochloric acid. In such a differential diagnosis becomes very difficult. There is no doubt that a certain proportion of gastric ulcers become carcinomatous, but my own experience does not at present support the high figures which have been given by MacCarty, namely, that 79 per cent. of cases of carcinoma show evidence of an ulcer. Of this present series, 6 cases were considered to be possibly carcinomatous even at operation, and therefore a partial gastrectomy was performed, rather than excision of the ulcer. In none of these, however, did the microscope show the presence of carcinoma. In 6 other cases, which are not included in the above list, a partial gastrectomy was performed owing to the doubtful appearance of the ulcer, and in all of these early carcinomatous changes were found in one edge of the ulcer. In 28 cases from the above list excision of an ulcer on the lesser curve was carried out, the condition being macroscopically a simple ulcer. In not one of these ulcers did microscopical section show the presence of any malignant changes. This difficulty is, however, of but little moment, for both conditions will require operative treatment, and should there be any doubt at operation a partial gastrectomy will be performed rather than excision of the ulcer, the former operation being, in my experience, not followed by a higher mortality.

Duodenal ulcer.—As a general rule, a duodenal ulcer may be distinguished by the fact that the pain is often more severe, appears at a later period after the ingestion of food, often wakes the patient at night, and is temporarily relieved by the taking of food or alkalies. The symptoms have even a more definite periodicity, vomiting is absent, and there is a more marked increase of the free hydrochloric acid in the test-meal. In some cases of gastric ulcer it has already been shown that the pain appears late after food, and occasionally, in them, the pain may actually be relieved by the taking of food. Ulcers just on the gastric side of the pylorus may occasionally wake the patient at night, but even in such circumstances vomiting is likely to be present, whereas with true duodenal ulcers it is very rarely seen.

Gall-stones.—In certain cases there may be considerable difficulty in making a diagnosis between gall-stones and gastric ulcer. If the stones have remained in the gall-bladder they will give rise only to gastric symptoms, but, as a rule, these can

be differentiated from those due to ulcer by the fact that they are much more persistent. Thus it is not uncommon to find that the patient may have suffered for 15 to 30 years with symptoms of this description, but it will nearly always be found that such symptoms have been present every day, there being no intervals of complete freedom such as are so characteristic when an ulcer is present. The pain is not nearly so severe and not uncommonly, provided that no complications be present, the patient will complain not so much of pain as of a feeling of discomfort. This feeling will have a definite relationship to food, and will come on much more rapidly than the pain of a gastric ulcer, being, as a general rule, noticed before the meal is finished. The discomfort will consist of a feeling of fulness and distension, so that the patient may have to loosen her clothes.

Sometimes, however, there will be, added to the above symptoms, attacks of more severe pain, which are probably dependent upon the presence of cholecystitis. These attacks of severe pain will appear at definite intervals, and may last only for a few days or weeks. The pain may be increased by taking food, but, as in the case of the feeling of distension, the increase of pain appears very shortly after the food is taken. Vomiting may also be present, but, as a general rule, does not relieve the pain so definitely as in the case of an ulcer. Should the history of the long-continued dyspepsia be overlooked a succession of these attacks may simulate gastric ulcer. There may, however, be definite tenderness over the gall-bladder, and this viscus may be distended and palpable. A test-meal may likewise be of value, for generally, when gall-stones are present, the free hydrochloric acid is absent, and the total acidity is low.

If there have been definite attacks of colic or jaundice the diagnosis is generally clear.

Appendix dyspepsia.—It is now clearly established that certain cases of chronic appendicitis will very closely simulate gastric or duodenal ulcer. As a general rule, the symptoms are less severe and tend to be more continuous, so that there may be several years' history of pain appearing a half to one hour after food, and being situated almost wholly in the epigastrium. Some of these may persist, with other gastric symptoms, for several years, and may then develop a definite attack of appendicitis, or there may be more severe attacks of dyspepsia, during which time the patient may have some deep or superficial tenderness in the appendix area. It occasionally happens, however, that the constant dyspepsia is so slight that but little attention is directed to it, and that exacerbations of pain occur which are almost limited to the epigastrium. In such cases gastric disease is more likely to be diagnosed, especially so as in some of these cases of appendicitis hæmatemesis may be present. A test-meal is not so satisfactory a guide as in some of the other conditions, for sometimes it may be raised; so much so that Sherren has recorded 7 cases in which the test-meal findings resembled those of duodenal ulcer.* This difficulty in diagnosis is increased by the fact that not infrequently such a lesion of the appendix is associated with an ulcer of the stomach. Hence, in all cases of gastric lesions, it is advisable that the condition of the appendix be investigated.

Treatment.

The evidence is becoming more and more clear that little or no permanent benefit can be obtained by the medical treatment of chronic ulcer of the stomach. During the first attack, while the ulcer is acute, it is advisable that a thorough course of medical treatment persisting for some two or three months should be tried. I have, however, shown that there is a certain amount of evidence that the majority of the cases of chronic gastric ulcer do not persist as such over the many years during which the symptoms have been present. It would rather seem that an ulcer is formed and persists over some months, that it then wholly or partially heals, only to relapse again at a later date, and such a relapse seems to be to a certain extent independent of medical treatment, for many of my own cases have undergone many months' course of medical treatment, and, although perhaps temporarily relieved, it has been no uncommon thing to find that a fresh acute attack would occur even while the medical treatment is being carried out. On the other hand, the success of surgical treatment is very definite. There are few lesions which have such uniform and rapid benefit from adequately planned surgical treatment. Not only is the actual ulcer rapidly cured, so that the sym-

* J. Sherren, 'Brit. Journ. of Surg.', vol. i, p. 396.

ptoms disappear within a few days of operation, but the tendency for recurrence appears to be overcome, so that with very few exceptions there is no further formation of an ulcer. In my own series of cases the results have been most gratifying. A large proportion of the patients inform me that they are absolutely free from all symptoms, and that they have never felt so well before. Of the total number two died as a result of operation. One of these was operated upon for an extremely severe hæmorrhage, so severe, in fact, that it was thought probable that the condition would prove fatal if operation were not undertaken. The other death occurred in a man, æt. 70 years, and was due to the onset of post-operative broncho-pneumonia. Of the remainder, final reports have not been received from 6 cases, leaving, therefore, 49 cases that have been followed since the time of operation. Of these, 37 are absolutely cured. They state they have no symptoms of any sort, that they have never been better in their lives, or that they are absolutely cured. Four of them have been followed for over 18 months, 11 for over a year, and 12 for over 6 months, leaving 10 who were operated upon less than 6 months ago. It is of interest, however, to note that among these latter several state that they are absolutely well even within a month of the operation. Two cases have recurred. One of them had at operation a large mass on the posterior surface of the stomach which was considered to be carcinomatous. A posterior gastro-enterostomy was performed to relieve the symptoms. He recovered completely and remained perfectly well for a year, when the symptoms returned. A second operation was then undertaken, and a well-defined chronic ulcer was found attached to the pancreas, the surrounding induration had disappeared, and the condition no longer resembled carcinoma. The ulcer was freed from the pancreas and excised, and the patient has remained perfectly well ever since, that is, for over a year. The second patient had an ulcer at the pylorus. He remained well for one year after operation, and then developed symptoms suggestive of gastro-jejunal ulcer. A second operation was performed, a gastro-jejunal ulcer found, the junction was excised, and a fresh one made. He has remained free from all symptoms since, a period of 5 months. The remaining 10 patients all express themselves as being better for the operation, but they all suffer from some minor symptom. Most frequently there are attacks of flatulence, especially after meals; occasionally there are slight attacks of vomiting, or the regurgitation of bitter fluid. One only has to give up work for short periods during the attacks. Several of the patients are now taking part in the war.

July 19th, 1915.

Pleural Effusion with Increased Vocal Fremitus.—S. Höyer reports the case of a married woman, æt. 49 years, who had a sarcoma of the uterus, for which hysterectomy was performed. About 9 months later she developed influenza, and the cough which followed was accompanied by profuse expectoration. There was slight fever, and over the base of the left lung numerous râles were audible; the percussion note was dull, and air entry was diminished. The dulness extended upwards a little beyond the spine of the scapula, and merged into the cardiac dulness in front. Vocal fremitus over this area was loud, and somewhat louder than on the right side. Exploratory puncture yielded slightly opaque fluid. A fortnight later the patient was very dyspnœic, and the percussion note was dull over the whole lung, the respiratory sounds of which were diminished. But vocal fremitus was still pronounced. The heart was displaced to the right, and the blood-stained fluid which was drawn from the left pleural cavity measured about 3 litres. Even after this aspiration, the dulness and diminished air entry persisted over the base of the lung.

The writer suggests that the fluid was distributed over the whole lung, as a shallow layer, which was insufficient to intercept the vocal fremitus conducted by the new growth to the chest wall. This paradoxical combination of increased vocal fremitus with pleural effusion may be useful in the diagnosis of malignant disease of the lungs.—*Norsk. Magazin for Lægevidenskaben*, p. 1297.

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We regret that owing to the war the number of pages must be diminished for a time.

A LECTURE ON FOREIGN BODIES IN THE ALIMENTARY TRACT.*

By W. McADAM ECCLES, M.S., F.R.C.S.,
Surgeon to the Hospital.

Gentlemen,—We are discussing this morning the subject of foreign bodies in the alimentary tract. We have had in the wards three cases illustrating the ingestion of foreign bodies. One was that of a child, æt. 1 year 8 months, who, a fortnight before it was admitted, swallowed a farthing, and the coin passed through the whole length of the alimentary tract, and was discharged at the anus. The child, not being satisfied with the farthing, then proceeded to swallow a French nail 1¾ in. long—a formidable foreign body for a child twenty months old (Fig. 1).

The second case was that of a boy, æt. 13 years, who, in play, put into his mouth a metal cylinder, test-tube like in shape, 2 in. long by ½ in. in diameter. This passed into his stomach and lodged there. I show you now the boy with the scar as the result of the operation. We had to take the cylinder out of the stomach. The operation being a comparatively recent one, the scar is red, and it is interesting to notice that the scar has become already a little stretched and somewhat keloid.

The third case was that of an infant, æt. 11 months, who swallowed a large ordinary dressmaker's pin. We have lost sight of that pin, and are not sure where it is; but it has caused the child absolutely no inconvenience. A skiagram has been taken but no evidence of the pin can be seen, nor has there been any evidence of its presence in the fæcal material which has been passed.

* Delivered at St. Bartholomew's Hospital.

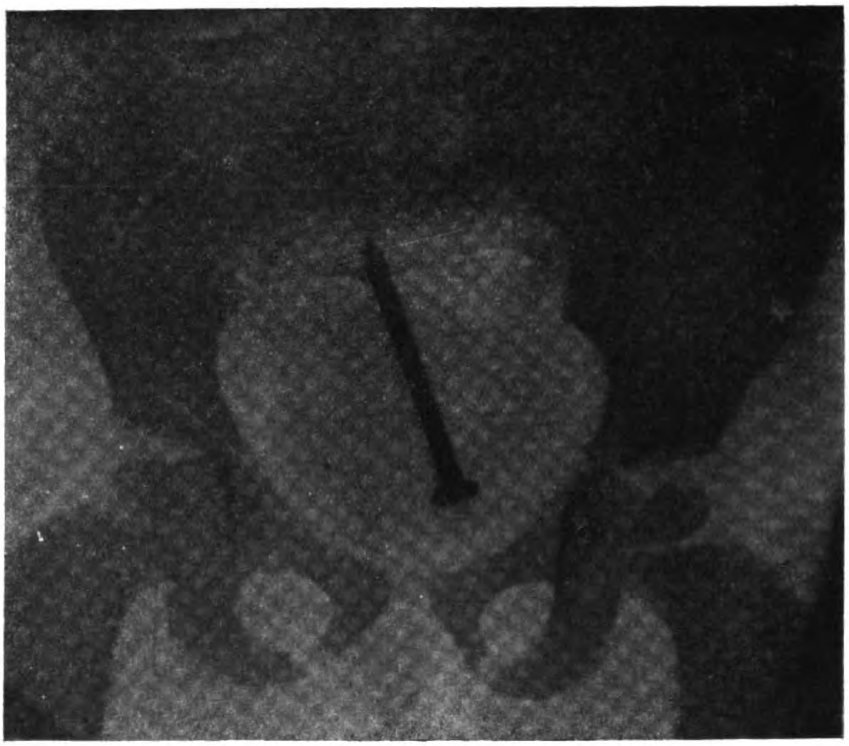

FIG. 1.—French nail in the rectum of an infant æt. 20 months.

Let me now say a word or two with regard to the anatomy of the alimentary tract in relation to foreign bodies. The first spot at which a foreign body is apt to lodge is in the mucous membrane of the inner surface of the cheeks. Here the broken end of a pipe-stem is sometimes seen. Then we come to a narrower portion at the lower part of the pharynx, and there quite a number of different foreign bodies may become lodged. The story goes of how, in a restaurant, in early days of new potatoes, a waiter was carrying a dishful to one of the frequenters of the place, and seeing one potato which he thought would be exceedingly nice, he put it into his mouth. Finding it very hot, he swallowed it, and it stuck just behind the thyroid cartilage, and caused complete obstruction for the time being, and of course, burned the mucous membrane, and as a result there developed subsequently a fibrous stricture of that region. Another interesting case was that of a man, who, for a wager, said he would swallow a billiard ball. He managed to get the ball in between his teeth—not at all an easy matter, and, giving an involuntary gulp, the billiard ball passed into the grip of the sphincter of the pharynx, with the result that he got not only obstruction to his alimentary passage, but also obstruction to his air passage, and before tracheotomy could be performed he died.

The œsophagus itself, because of its muscular tissue, is apt to grasp any foreign body which may pass into its lumen. You will recollect that the œsophagus at its lower end passes between the crura of the diaphragm, which act as a sphincter at that portion of the tube; and here, again, foreign bodies may become impacted.

When they have passed into the stomach, which is a wide cavity, there is a tendency for them to stick as they try to pass from the stomach into the intestine at the pylorus; at any rate, the pylorus contracts, and so prevents a foreign body from passing out of the stomach. It is interesting to remember that the so-called small intestine diminishes in calibre towards the ileo-cæcal region; in other words, the jejunum is greater in calibre than is the ileum. The actual ileo-cæcal valve is another spot where foreign bodies are likely to be retained.

In the large intestine itself foreign bodies are retained because of the sacculation between the longitudinal bands, and it is not an uncommon

thing to find a coin, for instance, which may have passed through the length of the small intestine, held up in the large intestine.

Lastly, we come to the rectum, a portion of the large intestine in which foreign bodies are by no means rare. They may reach it from the mouth, having traversed the whole length of the alimentary tract, or they may be introduced through the anus.

The first specimen I show you is a portion of a thin clay pipe-stem. A child, æt. 3 years, was trying to blow bubbles when it fell, and the pipe-stem ran into the child's cheek. The father, seeing the pipe sticking out of the cheek, dragged upon it, but did not realise that the stem was broken. This foreign body remained embedded in the cheek for almost a year.

Now, passing from the mouth to the œsophagus. I told you that the œsophagus is a part of the alimentary tract in which foreign bodies are very apt to lodge, particularly those foreign bodies which are irregular. And perhaps one of the commonest at the present day is the tooth-plate. Sometimes one of the sharp points of the tooth-plate rests against the wall of the œsophagus and ulcerates through into the thoracic aorta, which lies by its side, with the result that a violent hæmorrhage occurs and the patient dies in a very short space of time.

Such a plate may be swallowed during sleep, and frequently the owner does not know he has done so until he awakens in the morning, and the swallowing efforts tend to fix still further the plate in the œsophagus. These plates may be swallowed during anæsthesia, if the precaution is not taken to remove the teeth before commencing to give the anæsthetic. Sometimes the obstruction can be removed by means of œsophageal forceps, in which the blades only open at the extreme end, so that they can be manipulated in the very small space of the œsophagus. For coins in the œsophagus, or any other discoid structure such as a button, a coin-catcher is very useful. When this is passed down, it is slipped beyond the swallowed body, and, as it is withdrawn, the coin gets caught between the ring and the stem of the instrument. The catcher is flexible so that it can round curves in order to reach the œsophagus. Nowadays we have the great advantage of being able to perform this little operation of extracting coins from the œsophagus under X-rays; in other words, observing the coin and the coin-catcher on the screen and seeing the coin-catcher grasp the coin.

We now pass from the œsophagus down to the stomach. The stomach is a receptacle for a great variety of foreign bodies. I think the most interesting I have ever seen in the stomach was quite a large piece of jagged glass. How ever the patient, who happened to be a lunatic, managed to get it down to the stomach I cannot imagine. There was a picture hanging in the room where he was with some glass in the frame. He took this picture, smashed the glass, and swallowed a considerable number of pieces, including the one found in the stomach, which was 3 in. long and very sharp-pointed at one end, and nearly 2 in. broad at the other end.

In this skiagram you see the cylinder *in situ* in the stomach of the boy whom we saw. The cylinder is lying transversely. Another skiagram shows it even better, taken when the stomach tried to force the cylinder out into the intestine. We gave it a chance of being passed on, but it did not succeed in reaching the duodenum, and so we had to open the stomach and extract it.

In the stomach also may be found an extraordinary type of foreign body known as a hair-ball. These occur much more frequently in animals, especially oxen, because they lick their hides, and in that way hairs are carried into the stomach. When found in the human stomach, it is almost always in women, and generally in women who are hysterical, they bite their hair off and swallow it (Figs. 2 and 3).

I throw on the screen now, rather as a matter of history, a Murphy's button. It is a metal button; its use was one of the steps in the progress of anastomosis in the alimentary tract. Murphy introduced this button in order that the operation might be done rapidly, and, as he thought, with very great safety. When it was used for gastro-enterostomy it was apt to fall into the cavity of the stomach and remain there as a foreign body. I believe there are persons still going about with a Murphy's button in their stomach without being caused any inconvenience thereby. It may pass into the small intestine and cause obstruction there, or be lodged at the ileo-cæcal valve.

We pass now to the intestine. Some foreign bodies appearing in the intestine may be termed self-made, and are of great interest. These are gall-stones. Here is one in the jejunum. How

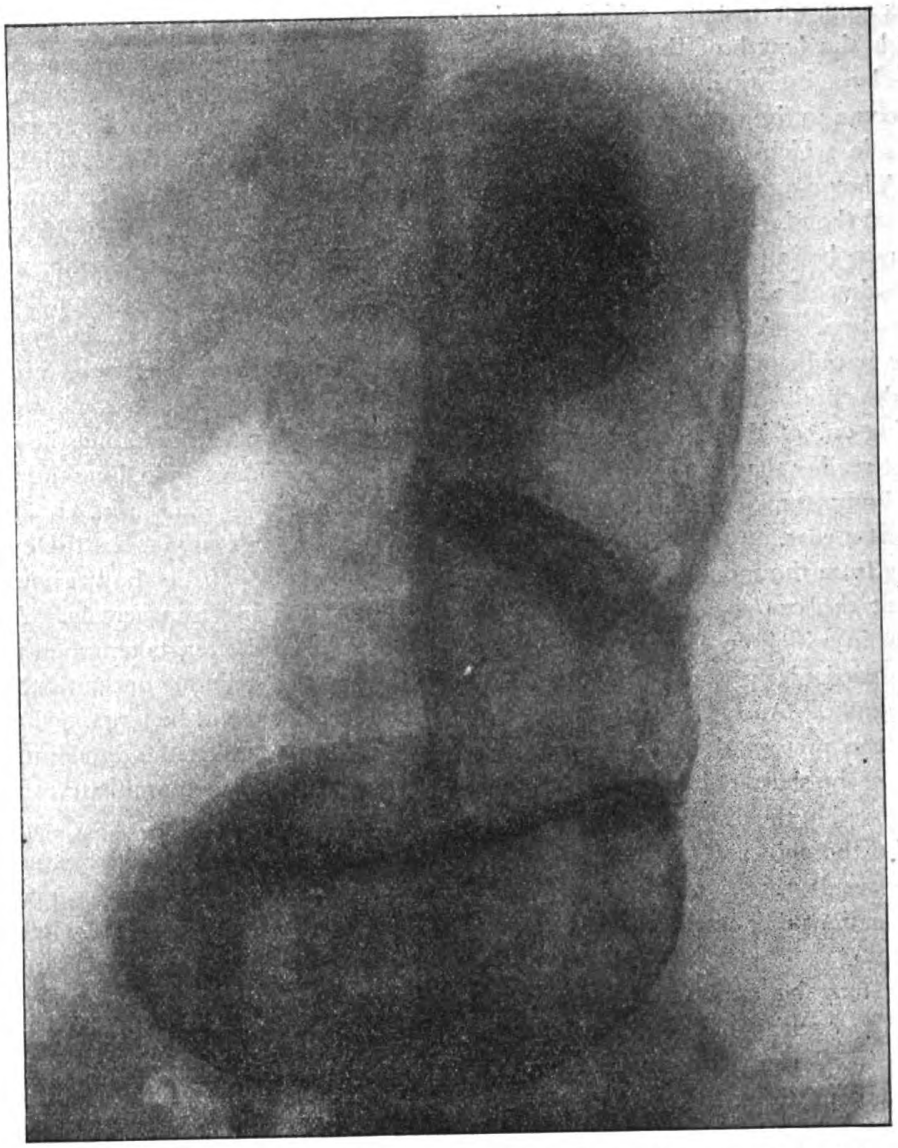

Fig. 2.—Skiagram of three hair-balls in the stomach. (Thurston Holland in 'Medical Annual,' 1914.)

has it got there? It was formed, of course, in the gall-bladder, which may contain enormous stones. They cannot pass along the common bile-duct to reach the duodenum, but the gall-bladder becomes adherent to the duodenum by a pericholangitis, and then ulceration occurs between the gall-bladder and the duodenum, and the stone passes into the small intestine through the opening thus formed.

These gall-stones may produce intestinal obstruction. I may give you the histories of three interesting cases. A gentleman, a year before I saw him, had a large carcinomatous growth of the rectum—so large was it, that the surgeon who saw him first said that before long he would require colostomy. The patient, however, went round the world after this advice, and when he came back he determined he would proceed on a motor tour round England. He got as far as Hindhead, and had lunch there. He then began to vomit. He said, "Probably the time has come for the colostomy to be done." I saw him six hours later. The abdomen was much distended, and he was vomiting repeatedly. He said, "Do not you think colostomy ought to be done?" and I said "Yes." One hour later I exposed the sigmoid through an incision in the left iliac region and found it about the size of my index finger, showing that it was not distended,

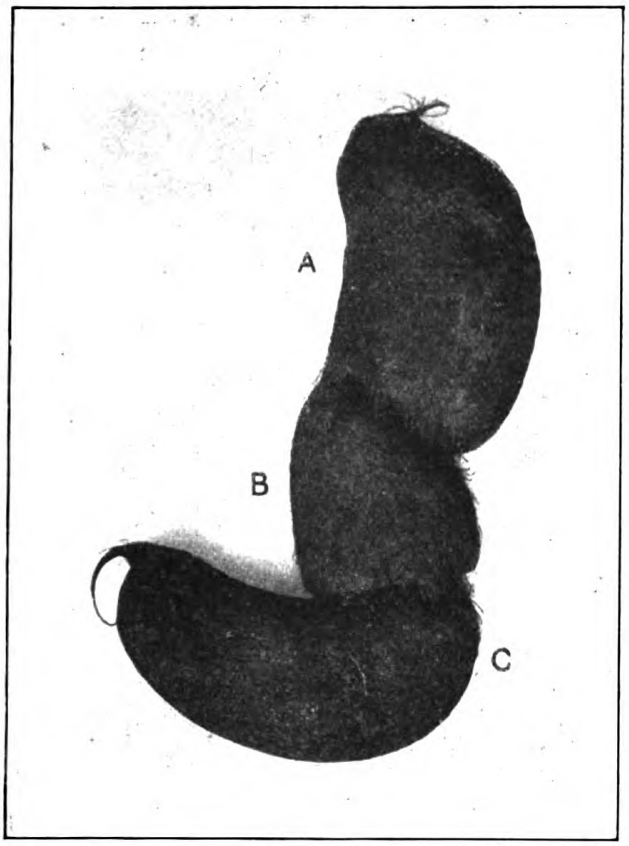

FIG. 3.—Photograph of the same three hair-balls after removal. (Thurstan Holland in 'Medical Annual,' 1914.)

but contracted. I therefore put my finger inside the wound, and felt in the abdomen a hard lump, which I took to be a secondary deposit causing obstruction of the small intestine. I pulled it out through the incision. It was a large gall-stone blocking the ileum. I excised it and sewed up the ileum, and the man lived for another year, when he died from secondary deposits, but without having had a colostomy done.

A woman, æt. 61 years, some eighteen months before, had a severe "gastric" attack. After this but little trouble, except flatulency, until one day she had an attack of vomiting with a little pain. Attacks of vomiting once or perhaps twice in the twenty-four hours continued for nine days, when, after a dose of calomel, vomiting became urgent and much more frequent.

When seen there was no evidence of distension, and the muscles were not tense. Percussion revealed a markedly tympanitic note over the right half, but some impairment of resonance on the left side.

The abdomen was opened as for a left iliac colostomy, and the pelvic colon was found not to be distended. On the hand being introduced a hard "lump" was felt, and on this being brought out of the wound it was found to be a large gall-stone tightly impacted in the lower part of the ileum. As the stone could not be shifted, a longitudinal incision was made over it, and it was easily removed. The intestinal aperture was closed with fine silk.

The vomiting was entirely relieved by the operation, but two complications caused a somewhat tardy recovery—a fairly smart melæna, and some foul smelling pus in her abdominal fat.

A man, æt. 79 years, who had been subject to "gastric" attacks, began to vomit on December 27th, and continued to do so once or twice in the twenty-four hours until December 31st, when for a period of forty-eight hours he had no vomiting at all.

On January 4th the vomiting recommenced, and was distinctly stercoraceous.

When seen on January 5th, he had a pulse-rate of 108, and feeble in character. Temperature was 98·8° F., and respiration 26. The abdomen was not distended, and the abdominal muscles not tense. Percussion revealed a tympanitic note over the right half, but less marked on the left side.

A diagnosis of obstruction by a gall-stone in the ileum was made. The abdomen was opened as for left iliac colostomy. The pelvic colon was found not to be distended. On the hand being introduced a hard "lump" was felt, and on this being brought out of the wound it was found to be a large gall-stone tightly impacted in the lower part of the ileum.

The manipulation caused the intestinal wall to crack slightly and transversely over the stone, so the rent was increased and the calculus extracted and the intestinal wound closed with fine silk.

The patient was entirely relieved of his vomiting, but he gradually sank and died thirty hours after the operation (Fig. 4).

Passing now to the large intestine, we come to the appendix. This little "back-water" of the large intestine is very apt to be a site for the lodgment of foreign bodies of all kinds, but perhaps pins and shots are the most common. I show you here a series of three specimens. The first is an ordinary dressmaker's pin, and it is interesting to

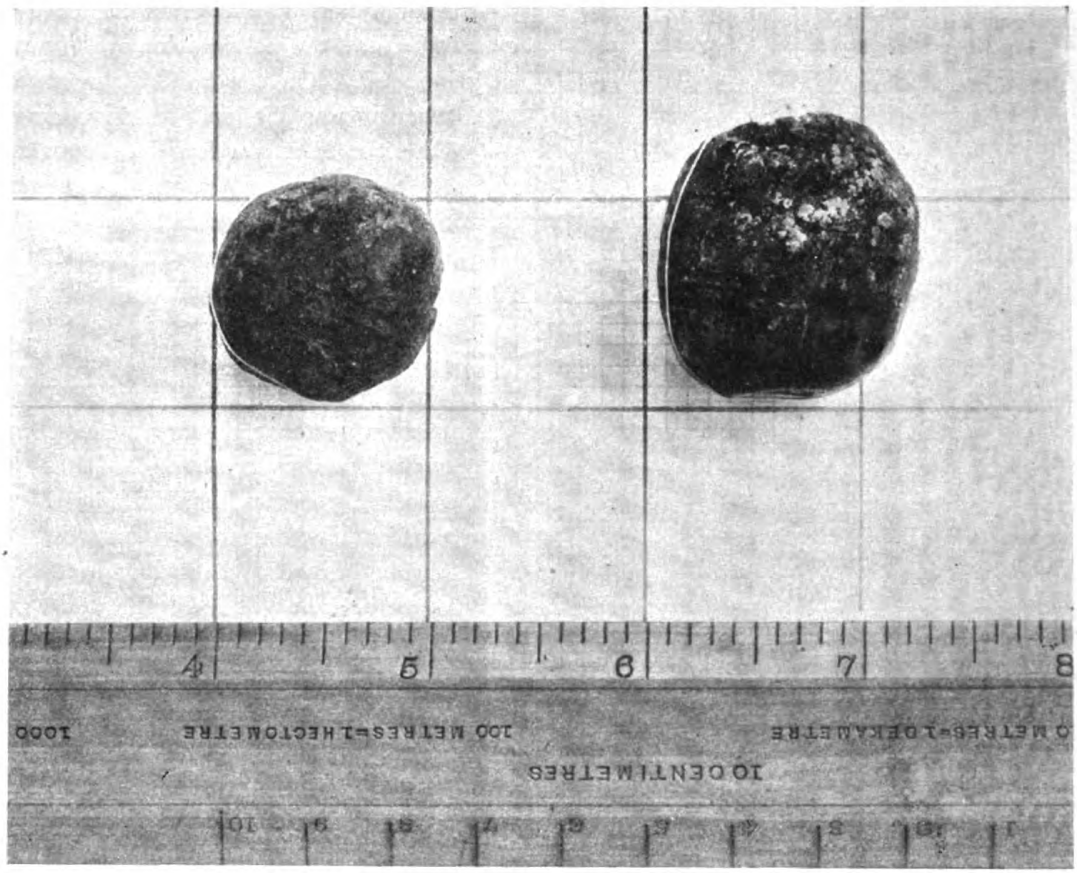

FIG. 4.—Two gall-stones which caused intestinal obstruction in the ileum. Photographed natural size.

reflect how the pin gets from the mouth along the difficult tract of the lower part of the pharynx, down the œsophagus with its numerous folds, into the stomach, then past the pylorus and round the curve of the duodenum through the flexure at the junction of the duodenum and jejunum, along the twenty feet of small intestine, through the ileo-cæcal valve, and then into the small aperture connecting the appendix with the cæcum. It is really an astonishing feat. A fact of great interest, moreover, is that in a certain number of cases one finds the "business" part of the pin is towards the distal extremity of the appendix. In other words, it must have entered the appendix with its sharp end forwards. Here is a very pretty specimen showing a pin *in situ*. The head of the pin is at the distal end in this case. The other specimen is a pin with a considerable concretion. One of the most interesting foreign bodies in the appendix I ever saw was in a soldier after the Boer War. He came to the hospital with all the symptoms of chronic appendicitis. We found an appendix about 6 in. long, and at its distal end there was a pear-shaped pellet of solder, evidently from a tin from which he had eaten beef in South Africa. Another very interesting form of foreign body in the appendix is a shot; sometimes you find a single shot at the blind end of the appendix causing considerable irritation and setting up all the signs of chronic appendicitis.

I told you that in the sacculæ of the large intestine coins are apt to get lodged, and here in the cæcal region you see a coin which has lodged in that position, and it remained there a considerable time. Here is another skiagram showing a metal mouth organ in the large intestine. Coins are swallowed very frequently by children in play, and not infrequently one sees coins in the intestines. It is interesting to note that there is considerable verdigris on a copper coin by the time it is passed by the anus.

Passing now to the rectum itself. We said that foreign bodies may pass the whole length of the intestines and reach the rectum, and the first one of interest which I have to show you is a French nail. It was swallowed by an infant of twenty

months. The first skiagram taken showed the nail in the upper part of the intestine. Comparing it with the breadth of the pelvis, you will see the nail is of very considerable size for a small child to have swallowed. Apparently it passed through the alimentary tract to the anus peacefully, for it did not worry the child in the least. Here is a tooth-plate, which passed through the whole length of the alimentary tract. I told you how frequently tooth-plates stick in the œsophagus, but this one went through and was voided *per anum*. This tooth-plate has not such sharp points as the other one.

If a child, other than a baby, swallows an irregular sharp body such as a small toy soldier, it should be given "stodgy" food—new buns, for instance—and it is well to mix up with the food some cotton-wool so that the foreign body may be clothed and rendered more smooth, and its passage along the alimentary track made easier.

Some years ago I had an interesting case, in which a lady, who had taken large and many doses of bismuth subnitrate for chronic indigestion, eventually had lodged within the second part of her rectum a "bismuth" ball, which caused intestinal obstruction.

A specimen or two will show foreign bodies introduced into the rectum through the anus. One of the most interesting we have in our Museum is this tobacco pipe. It was introduced by a man, while drunk, into his rectum, and it was not by any means an easy thing to get it out of the lower part of the alimentary tract.

The next specimen is still more extraordinary. It is one of those earthenware jars which I think is used for containing Liebig's extract of meat. It has a narrow neck and a comparatively small mouth. This patient suffered from intolerable itching at the anus—pruritis ani—and he found pressure effective in allaying this persistent irritation, so he hit upon the idea of putting a rounded object—a potato—into the mouth of the jar and sitting down upon it. But apparently he did not know that the sphincter was dilatable, and the continued pressure produced such a dilatation that the potato and the jar slipped up into the rectum. It was one of the most difficult foreign bodies to get out of the rectum, for the surface of the jar was exceedingly smooth and slippery, and therefore it required a considerable amount of dexterity to get it out.

July 26th, 1915.

CLINICAL JOTTINGS.

(*Continued from p.* 231.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

When Arterial Tension is High, it is not always well to lower it.

High blood-pressure is often spoken of as if it were a disease of itself requiring urgent treatment, whereas it is but a single symptom, and is often best left untreated. Its value in diagnosis and its call for treatment varies with the causes upon which it depends. A patient would be best without granular kidneys, but having that disease he should have a high arterial tension. If the tension be low he is not so well, and will not be better until the tension rises again to the adequate super-normal level. This explains why digitalis may do harm as well as good in granular kidney. If the tension be low, it will raise it and do good; if the tension be already high, it will do harm.

When arterial pressure is measured it is usually the systolic pressure which is determined. This depends chiefly upon the force of the ventricular contraction, and is held to be but little affected by the thickening of the wall of the artery. What it is far more important to know is the diastolic pressure—*i. e.* the more or less constant pressure maintained in the artery between the pulse-beats—and this must necessarily depend greatly upon the condition of the arterial walls.

In estimating these different conditions in the arteries and pulse, there is no apparatus yet invented which is in any way equal to the educated finger.

When the high tension occurs in the course of some general disease, such as granular kidney, the treatment will be in great measure that of the associated disease.

But high arterial tension may occur where there is no granular kidney nor any other obvious cause. The condition is not infrequent, and is met with for the most part in busy, over-busy, energetic, bustling men with much to do, and who do too much—men who work at high pressure. This common metaphor does, indeed, express the fact, for their blood-pressure is actually high. It is probably just

because it is high that they are capable of so much, and to reduce it means reduction in their capacity for work.

I have met with patients in whom some casual ailment, which brought them to the doctor, has been referred to this high tension. The high tension has been treated at first by mild drugs and measures, and, when these failed, by more drastic measures, stronger drugs, under-feeding or semi-starvation, and even free bleedings, with diastrous effect upon the general health and little or no effect upon the tension. In fact, the high tension in these cases, though no doubt in itself undesirable, seems to be the normal condition for that individual which alone enables him to retain his mental and physical vigour, for he loses both as soon as the pressure is reduced.

Thus it follows that there are cases of high arterial tension which are best left untreated. This statement is contrary to much of the generally accepted teaching of the day, but it has been brought home to me forcibly and not infrequently.

The Shape of the Thorax due to Muscular Action.

The rounded shape of the normal chest is due to muscular action. It is the position of equilibrium between the pull of the opponent muscles, those which expand and those which contract the chest.

This is proved by the peculiar change in shape which occurs in some cases of hemiplegia, for the paralysis may affect the muscles of the thorax, just as it does those of the face and extremities (*cf.* West, 'Diseases of the Respiratory Organs,' vol. ii, p. 658). Then, as the patient lies upon the back in bed, the affected side becomes curiously flattened.

A similar explanation may apply to the flattening which is observed after acute pleurisy or pneumonia. This flattening is often referred to adhesions which have formed between the two layers of the pleuræ. But it is too marked to have developed in this way, and occurs too soon after the attack, and, again, it disappears too quickly during convalescence. Where the pleura is adherent, of course flattening would be produced, but it would last longer and be more difficult to cure. The true explanation would be that the inflammation has spread from the pleura to the intercostal muscles. And just as in the early stage they are inhibited and do not move, so in the later stage they become directly involved, and do not act in the normal way again until some time after the acute inflammation is past.

The flattening of the side which follows ordinary pleurisy or broncho-pneumonia is best seen from the back, when the patient is sitting or standing up. It may be very obvious then when it is not so easy to detect in the recumbent position from the front.

(*To be continued.*)

Idiosyncrasy to Aspirin.—Mathé reports the case of a man who had long suffered from malaria, and whose liver was inactive, had recently been in hospital for typhoid fever. He had shown intolerance to various drugs. Thus, quinine in doses of 1½ gr. caused malaise, and he had suffered from cardiac symptoms, requiring medical attendance after a cachet of pyramidon; while on another occasion he had been very ill after a dose of antipyrin. When suffering from intercostal neuralgia, which lasted 24 hours and caused sharp pain on respiration, he took a cachet containing 7½ gr. of aspirin. Twenty minutes later the body became covered with an eruption, the face was red and swollen, the eyes congested, the lids œdematous, and his features became grotesquely changed in a few minutes so as to resemble a person suffering from severe facial erysipelas. The nose was swollen and the patient complained of a sensation of dryness which prevented him breathing through it, while swelling of the mouth and throat was accompanied by a choking sensation. The abdomen also became distended. These symptoms persisted until he was seen by the writer, who at once applied a mustard plaster to the feet. In a few minutes the nostrils became moist, and the sensation of respiratory distress disappeared. The other symptoms also rapidly improved, and in a few hours he had recovered, with the exception of the eruption on the body and congestion of the face, traces of which persisted for 24 hours.—*Bull. de la Soc. de Méd. de Paris*, p. 170.

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We regret that owing to the war the number of pages must be diminished for a time.

THE EFFECTS OF THE POISONOUS GASES USED BY THE GERMANS.*

By R. MURRAY LESLIE, M.D., M.R.C.P.,
Senior Physician, Prince of Wales' Hospital; Physician, Royal Hospital for Diseases of the Chest; Civil Physician, Hampstead Military Hospital.

NATURE OF THE GASES.

Professor J. S. Haldane, F.R.S., the well-known expert, is of opinion that the asphyxiating gases hitherto used by the Germans are mainly chlorine or bromine. The former is readily generated from bleaching powder and is of a yellowish-green colour, and is two and a half times heavier than air. Bromine is still heavier and is of a yellowish-red colour, and is obtained from the mother liquors in the Stassfurt Potash Works. The gas is projected from cylinder tubes and appears in the form of a yellowish-brown smoke, which floats slowly along with the wind close to the ground and is readily visible for a considerable distance. On May 25th the gas-cloud rose in places to a height of forty feet over a five mile front.

THE EFFECTS OF THE GASES.

The main effect is to produce an acute irritation of the respiratory mucous membrane, resulting in an outpouring of thin muco-serous fluid which threatens to drown the patient in his own secretions. Probably there is also some primary asphyxia due to spasm of the glottis. The intenseness of the irritation may be judged by the fact that not only is there smarting of the eyes, vomiting, violent coughing, and choking, but the soldiers' buttons turn green and their bayonets black when the gas reaches the trenches comparatively undiluted. After death tenacious, frothy fluid is found in all parts of the lungs, filling up the trachea and the large and small bronchial tubes.

* Lecture delivered at the Incorporated Institute of Hygiene.

Even when cyanosis begins to disappear the respiration continues hurried and panting, and it is believed by some that an acid condition of the blood is produced (acidosis). This point is now being investigated by Mr. Barcroft, of Cambridge. As far as his investigations have gone they tend to support the theory that the panting respiration may be of a specific nature.

Some military surgeons have stated that gas poisoning has given us the worst phases of suffering caused by the war; so much so that at casualty field stations many men were actually rolling in agony and praying to be allowed to die so as to be put out of their misery. Not a few were killed outright in the field by the asphyxiating gases, and Captain Bertram wrote to the 'Lancet' that he saw with his own eyes on one small stretch of road twenty-four unwounded men lying dead from the effects of the gas.

Resemblances to Pure Chlorine Poisoning.

In the rare cases of acute chlorine poisoning occurring in chemical works the symptoms are similar to those produced by the German gases. There are the same intense fits of coughing, with asphyxia, leading frequently to unconsciousness. These symptoms may grow worse and worse until death ensues in from twenty-four to forty-eight hours.

Degrees of Gas Poisoning ("Gassing").

The severity of the cases depends on three factors: (*a*) The *concentration* of the gas (whether encountered undiluted or diluted with air), (*b*) the *duration of exposure*, and (*c*) the *efficiency of the respirators* and the care with which they have been applied.

For general purposes the cases have been divided into three groups: (1) Mild cases, (2) moderately severe cases, and (3) very severe cases.

(1) *Mild cases.*—The men belonging to this group have smarting and pain in the eyes, headache, occasional vomiting, frequent painful cough, and in some cases greenish viscid expectoration. There is generally distaste for food, which is nauseous and tastes of gas. There is, however, no definite cyanosis or asphyxia, and the symptoms tend to disappear rapidly, with the exception of frequent respiration, which lasts for some days.

(2) *Moderately severe cases.*—In the moderately severe group the patients are definitely ill, with severe headache, vomiting, cyanosis, quick panting and painful breathing of a strangling nature, causing acute discomfort. They are often drowsy, and the symptoms tend to get much worse at night. There is generally some fever, the temperature rising to about 100° F. Bronchitis is a common sequela.

(3) *Very severe cases.*—In the very severe group there is the most urgent dyspnœa and marked asphyxia. The faces are blue-black, and the patients suffer agonies, with rapid pulse, gasping or strangling breathing, constant coughing, and hoarse rattling voices. Semi-consciousness often supervenes and may pass on to unconsciousness and death. In one such case the surgeon resorted to bleeding, and the blood removed was intensely dark, and clotted with remarkable rapidity; no relief being obtained, oxygen was administered without benefit, and this patient died within forty-eight hours from the time of being exposed to the fumes. Broncho-pneumonia and even pulmonary gangrene are occasional sequelæ.

Stages of Gas Poisoning.

Gassing and its effects have been divided into three stages: (1) a primary asphyxia, due to the irrespiratory nature of the gas, and partly to spasm of the glottis; (2) secondary asphyxia, due to the outpouring of fluid into the bronchial tubes; and (3) acute bronchitis and broncho-pneumonia.

I have already described the symptoms of the first two stages in referring to the degrees of severity. It is of patients in the third stage that we have had most experience at home. The majority of cases that have reached this country have exhibited symptoms of a mild bronchitis, but there has been a certain proportion of cases of severe bronchitis with broncho-pneumonia, and several have proved fatal. I recently saw a severe case of acute capillary bronchitis and broncho-pneumonia at one of the London military hospitals. The patient had the appearance of a man suffering from an attack of asthma. He was propped up with pillows, breathing about fifty respirations per minute, pulse rapid, and temperature up to 102° F. His voice was somewhat husky and speech was a distinct effort. There was not a square inch of his lungs over which rhonchi and crepitations could not be heard, and in certain areas there was evidence of patchy pneumonia. The expectoration

was no longer watery and frothy, but had become yellow and purulent. However, he did well.

I have a gas case with mild bronchitis now under my care who still has periodic attacks of burning pain referred to the mid-sternal region.

In the 'Lancet' of June 19th is given an important report to the Académie de Médecine of Paris, by Dr. R. D. de la Riviere, of the Institut Pasteur, and Prof. J. Leclercq, of Lille, on the effects of the German gases. They had under observation in hospital at Calais 112 soldiers who had been exposed to gas at Langhemarck. As the distance from the fighting line is short they were able to examine the patients a few hours after the intoxication. The irritation of the respiratory mucous membrane caused abundant expectoration, which was soon tinged with blood. Those who could not fly died vomiting blood. Others, much prostrated, crawled to the rear vomiting and expectorating blood, and passed bloody urine. On admission to hospital the eyes were watering and the eyelids were swollen. The cheeks and lips were violaceous, the features were drawn and the nose was pinched. The patients were harassed by dyspnœa and incessant cough. Some complained of a stitch in the side. There was abundant rosy frothy expectoration, sometimes bloody. Speech was painful and jerky. The majority of the patients presented symptoms of inflammation of the whole respiratory tract extending to the finest bronchial tubes. In these the condition was not grave. But in others the pulmonary lesion was severe and took the form of broncho-pneumonia or gangrene of the lungs. Two patients presented the phenomena of hæmolytic jaundice. Another showed hæmoglobinuria for several days. Several had persistent albuminuria. Most of the patients passed concentrated high-coloured urine containing much biliary pigment. At the onset the sputum was characterised by desquamative elements and some polynuclear cells, but soon it was modified so as to indicate congestion and, in some cases, gangrene of the lung.

Treatment.

The cases of bronchitis are treated in our hospitals by the ordinary methods (*e. g.*, moist antiseptic inhalations), and the same applies to broncho-pneumonia. It is during the first stages that special measures are required. In the very severe cases treatment is often of little avail. Bleeding, followed by transfusion with normal saline solution, has been tried, while oxygen* has been administered from cylinders. The asphyxia is largely due to lack of oxygen, and it has even been recommended to inject oxygen under the skin—a method which has been found useful in not a few cases. It is recommended that where possible patients should be treated in the open air, that the body should be kept warm, and that very little food should be given. The nausea and distaste for food render this last method easy to carry out. It has been recommended also to spray the air with adrenalin to allay the swelling of the lining of the bronchial tubes, and it has even been suggested that the adrenalin might be applied directly to the bronchial tubes.

In mild cases a slight bronchial sedative and plenty of fresh air is all that is required. In the rather more severe type of case belladonna or atropine, with the view of diminishing the profuse bronchial secretion, has been recommended, while emetics have also been found useful.

Mr. Henry Brunner, the well-known chemist, suggests the inhalation of alcohol by means of a cloth moistened with whisky, brandy, rum, or rectified spirit. It is known that alcohol absorbs chlorine with avidity, and the compounds formed by the interaction of the two substances, viz. chloroform, chloric ether, chloral hydrate, etc., are practically harmless. It is doubtful, however, if such substances would be formed under these conditions. Mr. Brunner's son, who was once accidentally gassed by chlorine, found the inhalation of Eau de Cologne extremely soothing. He considered the treatment by alcohol merely palliative, and unlikely to have any real remedial effect in serious cases.

Preventive Treatment.

This is far the most important aspect of the question. Gassing may be prevented in one of two ways:

(1) By *preventing the access* of the gases, *e. g.* by precluding their entrance into trenches or dwelling-houses.

* In the French official report, dated July 10th, is mentioned the capture at La Fontanelle of an oxygen apparatus intended by the Germans for use against the effects of poison gas.

(2) By *neutralising the poisonous gases* before they are breathed into the lungs.

(1) *Prevention of access.*—(*a*) In the trenches, gases can be used only when the direction of the wind is favourable and any change in the direction at once blows the gases back towards the trenches from which they have been projected. Special precautions will, therefore, have to be taken by the army on the windward side. The dilution of the gas is more or less directly proportionate to the distance traversed. By the time the gas is twenty feet away from the discharged cylinder, the ratio, according to Sir Hiram Maxim, is one volume of chlorine to five volumes of air. If a soldier were to breathe a chlorine mixture of even one in twenty he would not live a few minutes, but by the time the fumes have reached our trenches the dilution is about one in a hundred. Sir Hiram Maxim is stated to have completed an invention which, he claims, will force the poisonous fumes over the heads of the men in the trenches. It would be a great matter if this invention were to succeed in actual warfare, as, apart from its military effects, it would save an immense amount of suffering.

(*b*) *In dwelling-houses.*—In the warning issued by the Commissioner of the Metropolitan Police, it is recommended that: "All windows and doors in the lower floor should be closed to prevent the admission of noxious gases." The dropping of a bomb containing asphyxiating gases will be followed by a peculiar irritating odour and a smarting of the eyes, which symptoms ought to at once act as a warning. After having fastened the doors and windows of the lower floor, one should then betake oneself to the top rooms of the house.

(2) *Neutralisation of the poisons.*—It has been found that most of the asphyxiating gases are acid, and that therefore they can generally be neutralised by the use of an alkali. Common washing soda is the particular alkali that is found in every house. A 10 per cent. solution of washing soda, *i. e.*, 1 lb. to a gallon of water, or ¼ lb. to the quart, is about the right proportion, though the solution cannot be too strong and may be used saturated. An ordinary bath towel soaked in this lotion and in one or two layers will enable a patient to breathe in an atmosphere contaminated either by chlorine, bromine, nitrogen oxides, or sulphurous acid gas. It is well, however, to have a simple and handy form of respirator ready for use, and the Commissioner of Police in his recent warning advises a pad of cotton-waste saturated with a strong solution of washing soda and contained in gauze, which may be tied round the head. The pad should be large enough to protect the nose as well as the mouth, the gauze being so adjusted as to protect the eyes. Such a respirator can be made for a very trifling cost, and should be damped when required for use. The German soldiers have been equipped with respirators containing sodium hypo-sulphite, which neutralises either chlorine or bromine, and solutions can be readily obtained containing both carbonate and hyposulphite of soda in certain proportions. The 'Chemist and Druggist' on June 5th recommends a formula in which the proportion is 15 oz. of hyposulphite and 5 oz. of sodium carbonate. The official War Office solution is supposed to be 4 oz. of carbonate of soda and 6 oz. of hyposulphite of soda to half a pint of water. The French Academy of Medicine recommends the following solution: Sodium hypo-sulphite, 1000 grm.; sodium carbonate, 200; glycerine, 150; and water, 800.

The hyposulphite is now getting very expensive, while carbonate of soda, on the other hand, is exceedingly cheap and probably quite efficacious without the hyposulphite, and anybody can dissolve a pound in a gallon or a quarter of a pound in a quart bottle. Sodium bicarbonate may be used instead of the carbonate, but tends to be somewhat irritating to the skin, which is not the case with sodium carbonate, even in strong solution. The chlorine unites with the soda, forming sodium chloride, or common salt, which is harmless. A portable respirator can be made by saturating the wool or the flannel or the cotton waste with a strong solution and allowing the respirator to dry. The dried respirator can then be carried in the pocket and damped with ordinary water before use. A strong solution should be used, the respirator being soaked in it and the excess of the liquid squeezed out. If kept in a sponge bag it will keep moist for many days. No one should go to bed without knowing where the respirator is, and if the latter is dry, without knowing where the water to damp it with can be obtained without a moment's delay. It should be at one's bedside. It would probably be an exceedingly good plan for every household to have a private Zeppelin rehearsal, so as to see how quickly the basement

windows can be closed, the respirators affixed, and the members of the household collected on the top floor. On May 24th certain battalions escaped practically unscathed owing to the fact that their Commanders, warned by the gas surprise at Ypres a month earlier, had initiated respirator drill and given their men the most stringent instructions about keeping the respirators moistened.

Various patterns of respirators can be obtained. The official one consists of cotton-waste soaked in lotion and covered with black gauze netting, which reaches over the whole face so as to cover the eyes. The Institute of Hygiene respirator consists of a combination of cotton-wool, layers of flannel, and towelling, with a separate face-piece of black net, which covers the whole of the face, including the respirator. The fastenings permit of the respirator being drawn down under the chin until required, and it can be slipped on to the mouth in a second, covering the nostrils. The black net is specially intended for the protection of the eyes, and may be drawn up on the forehead ready for use, and can be instantly pulled down over the chin. Other respirators, like the Cantlie respirator, are practically masks, with pieces of talc over the eyes, which protect the eyes, at the same time allowing clear vision.

August 2nd, 1915.

A simple means of destroying Pediculus Vestimentorum.—Jousseaume states that the parasites may be induced to shift their quarters by introducing the bowl of one's pipe under the vest, so as to fumigate it thoroughly with tobacco smoke. If the garment can be removed the fumigation will be more efficient. This parasite being the transmitter of typhus and relapsing fever its destruction is important for the health of the army. Troops might be supplied with rubber tubing from 10 to 20 inches long, with which they could fumigate their own or comrades' clothing during periods of leisure. This simple means does not supersede the more thorough disinfection of the soldiers' clothing by heat when this can be carried out, but is a speedy method of freeing oneself from a pest of active service. Spreading lice-infected garments on an ant-hill will also rapidly free them from parasites.—*Bulletins de l'Académie de Méd.*, p. 48.

PROLAPSUS UTERI.*

By THOMAS HOLMES, M.D. M.S.,Lond.,
Surgeon to the Cheltenham General Hospital.

AMONG the cases we have just seen in the wards are several of uterine displacement which have been operated on. In order to understand the mechanism of prolapse, it is necessary to understand the physics of the pelvis. A ring of loose tissue begins at the retro-pubic fat in front, passes on the internal aspect of the obturator internus, and the upper part of the levator ani on the left side, then between the posterior vaginal and anterior rectal walls, then on the inner aspect of the obturator internus and levator ani on the right side, and so back to the retro-pubic fat.

By this ring, the pelvic floor is divided into two portions: an entire displaceable portion, and an entire fixed portion. The entire displaceable portion is composed of the bladder, urethra, and vaginal walls, and has resting on it the uterus, tubes, ovaries, and broad ligaments.

The entire fixed portion is composed of in front the posterior aspect of the pubes sloping downwards and backwards, at the sides the inner aspects of obturator internus sloping downwards and inwards, and behind the anterior rectal walls sloping downwards and forwards. The funnel thus formed supports the entire displaceable portion, and these two combined form the pelvic floor, on which is supported the uterus, adnexa, and the abdominal viscera.

One great function demanded of the female pelvic floor is the support of intra-abdominal pressure; the abdominal and pelvic viscera, along with the bladder and vagina, rest on the entire fixed portion, which is enabled to support its burden by the inward convergence of its walls.

"Prolapsus uteri" is therefore not a mere uterine descent, but a downward displacement of the abdominal and pelvic viscera and the entire displaceable portion of the pelvic floor.

What is its ætiology?

There are three chief factors producing "prolapsus." A deficient support by entire fixed portion; through parturition the sacral segment has

* A Clinical Lecture given at a Demonstration at the General Hospital, Cheltenham.

become straightened out, or deficient in its lower margin, the perinæum, and also the slope of the levators ani has been lessened, or they have been torn. On the one hand, however, one can have a complete rupture of the perinæum without any prolapse, and a prolapse without any tear of the perinæum; in these cases the other factors I am about to mention come powerfully into play. Tear of the perinæum, however, diminishes sacral support, and if deficient levator ani support is also present, intra-abdominal pressure causes prolapse.

The second factor is deficient tone of the entire displaceable segment, and slackening of the loose tissue around it; this one regards as a very important factor, and I consider frequently seen, generally due to over-stretching at parturition.

The last cause we have to take into account is intra-abdominal pressure.

Prolapsus uteri is therefore really a hernia, with a definite sac of peritoneum, a definite canal to travel down, and definite coverings.

The symptoms are varied. I shall quote a description from my case-book, given me by the patient herself, in a bad case.

"Complains of weakness down below; discharge and soreness; some bearing down; not much pain; but some backache, and a slight show of blood at times—she was well beyond the menopause—also complains that she has to get up at least once in the night to pass water, and has also some inability to hold the same." This patient had most of the symptoms which one meets with in individual cases, but sometimes a patient complains of a weight and bearing down only, and sometimes they identify their trouble and say their womb comes down.

Physical signs.—I generally examine patients in the Sims position, but in some cases in order to estimate the amount of displacement, it will be found necessary to have the patient in the lithotomy position or even upright. On the patient straining, the anterior vaginal wall is found to bulge, and then pass out at the orifice, the uterus is displaced downwards, and lies with its axis coinciding with that part of the pelvic curve in which it happens to be, the posterior vaginal wall bulges forwards.

In complete prolapse, in addition to the uterus, the whole anterior vaginal wall comes outside the vulva, and the posterior vaginal wall is everted. Furthermore, through congestion the uterus becomes hypertrophied and the cervix elongated.

If an erosion or a split cervix is present the tissues all become sodden and œdematous, and a superficial necrosis or a clinal ulceration causing discharge of muco-pus and serum frequently supervenes; if in addition the cervix is chafed, hæmorrhage may occur, thus simulating malignancy.

If the displaced organs are replaced, the posterior vaginal wall reduces first, then the uterus, and lastly the pubic segment, on straining the prolapse occurs in the reverse order.

Lastly, as to treatment. In the first place we have seen how increased abdominal pressure forces down the movable portion of the pelvic floor; anything, therefore, which tends to increase the normal pressure should be treated, *e.g.*, a bad cough, or constipation. Also, muscular strains should be avoided, for instance, lifting heavy weights. I have more than once seen severe prolapse caused by lifting a heavy weight or by a sudden slip whilst carrying a weight. A young housemaid slipped whilst carrying a bucket, and the sudden strain drove the uterus down until the cervix was half through the unruptured hymeneal orifice; in another case a patient, who was some seven months pregnant, sent for me for sudden hæmorrhage. I found the cervix protruding from the vulva, the os patulous. The membranes could be seen like a small, dark watch-glass appearing through the patulous cervical canal, from which blood was oozing; this was entirely due to carrying a heavy weight.

Secondly, as to pessary treatment. If I use a pessary I use one either of tin, vulcanite, or celluloid. I find that a Hodge, or an Albert Thomas, or a plain ring are applicable to most cases, and may be used in slight cases. In cases of complete procidentia, if the patient cannot stand a radical operation, a cup and stem pessary will be of service, or occasionally a plug of antiseptic wool frequently changed will keep the uterus in the vagina if it is reducible. The rubber-covered or glycerine-filled pessary invariably become septic, and unless great care and cleanliness is used are positively harmful. A daily douche ought to be used, and the pessary taken out and cleansed with soap and water and weak lysol frequently.

Thirdly, as to operative treatment. As we have seen from the anatomy and pathology of the subject, the matter is a complex one. The uterus

wants raising and maintaining at a higher level, the canal down which it has travelled has to be contracted, and often the bulk of the uterus itself has to be lessened.

Then, again, it is important to recognise whether the patient's symptoms are due to a "uterine prolapse" or whether there is, as often happens, a condition of general visceroptosis, with a slack abdominal wall, prolapse of the kidneys, and marked visceral prolapse. In a case like this I do not believe in operating on the uterine condition alone, if at all. In considering the operation to be done, it is also important to consider the age of the patient and the condition of the abdominal wall, as with feebly-developed and possibly separated recti and a fat, flabby abdominal wall the abdominal wall cannot be made use of as a basis for support of the uterus.

Some cases from my note-books will illustrate the subject.

Miss S—, æt. 55 years, multipara; on examination, cervix protrudes from the vulva about 1 in., is split, eroded, and ulcerated; on reduction, cystocele found, and perinæum reduced to a strip, about ⅛ in. broad, of scar tissue resting on the external sphincter; uterus bulky and œdematous.

Patient refused any abdominal operation, but consented to a vaginal one, as her life was miserable and no pessary relieved her. After a rest in bed, with douches, etc., the uterus was curetted, the cervix amputated, a large oval piece of mucous membrane removed from the base of the cystocele, and a colpoperineorrhaphy was done. Four years afterwards the patient was practically in the same comfortable condition that she was when she left the Home. There was no sign of prolapse of the uterus, and the only complaint was that of a little chafing at the vestibule occasionally.

Mrs. T—, æt. 50 years. Slight prolapse, very marked cystocele, causing tenesmus of bladder; perineal body quite destroyed. Anterior and posterior colporrhaphy done, and perineorrhaphy; when seen some years after, keeping quite comfortable and without any discomfort.

Mrs. W—, æt. 42 years. Uterus bulky and prolapsed cervix down to vaginal orifice; morbid cystocele and rectocele; pessary treatment gave no relief; prevented from following business, and unable to walk about without great discomfort.

Again, uterus curetted, anterior and posterior colporrhaphy done. After operation quite relieved and emigrated to Canada, where I heard that she was in excellent health and was nursing a chronic invalid, involving heavy strain in moving him about.

I have quoted enough of these cases to show that how, even in severe cases, very great alleviation of the condition can be obtained, and, what is more important from some patients' point of view, pessary life can be ended.

Another vaginal operation which I have not performed myself, but at which I have often assisted, is vaginal fixation, which is highly thought of by some authorities; but as I have not performed the operation myself I will not pursue the matter.

And now I may come to the question of operations involving the opening of, or section of the abdominal wall.

Those that I have practised have been suspension either by the round ligaments or by direct connection with the abdominal wall.

Though I have shown you to-day a case of shortening of the round ligaments intraperitoneally for retroflexion of a uterus which was bound down by firm peritoneal adhesions in the pouch of Douglas, and which first had to be freed, I should only adopt the same plan in prolapse when the vaginal canal had been first operated on, and where a great amount of traction was not to be expected. I consider that passing the round ligaments through the broad ligaments and suturing them at the back of the uterus, and so forming a sort of sling, will give much more satisfaction, and this was done in the other case I have shown you, where prolapse was severe, the cervix protruding outside the vulva, and the patient was quite incapacitated for work. Now one finds on examination that the cervix is high up in the vagina, and, as there was very little tendency to cystocele and rectocele, I did not think it necessary to do any vaginal operation. If she complains at all later on I should do colporrhaphy; but the trouble with her was more due to lack of tone and stretching of the levator ani and loose tissues than lower down, for her perinæum is quite intact. I did intend in her to attach the uterus to the abdominal wall by the filigree method, but this was so infiltrated with fat that I expected trouble with my filigree if I put it in, and so abandoned the idea.

In two cases lately I have used a filigree similar to the one I have here. In this operation the filigree is attached to the back of the uterus and then interpolated in the abdominal wall extra-peritoneally. In both these cases there was partial procidentia; but so far the result has been very satisfactory, the abdominal wall being a firm one, and the patient being very comfortable.

Ventrofixation may also be brought about by direct union of the uterine wall with the anterior abdominal wall subperitoneally, and this may bring about a very firm union.

In attaching the uterus to the abdominal wall I always obtain permission to sterilise the patient if of child-bearing age. I may now quote a case to show what may happen if pregnancy occurs.

The case was one which some years ago I was asked to see in consultation as the patient had an abdominal scar, was pregnant, and the doctor was puzzled with the shape of the abdomen.

I found that although she was seven months pregnant all the enlargement was below the umbilicus and was transverse in direction, the fœtal head lying to the right and the breech to the left, and that the uterus was firmly fixed to the abdominal scar. I foresaw trouble at birth and recommended abdominal section and Cæsarean, but we could not get consent. At full time I was again called in, an abdominal operation was positively refused, so we had to deliver by the vagina. This was effected by podalic version with very great difficulty, as the cervical canal was found to be long and irregular, and had to be dilated up by the hand, and then the head was found lying in a pouch on the right side and the body in a larger pouch on the left side. Powerful traction was necessary, and though fortunately the patient recovered quite normally, we were much afraid the uterus would rupture.

In complete procidentia it depends very much on the general condition of the patient as to what can be done. It often occurs in elderly women suffering from a chronic cough, and very bad subjects for operation. Occasionally, however, one comes across a case that is operable, and the choice then lies between colpoperineorrhaphy combined with either some method of suspension from above, or hysterectomy. Hysterectomy alone in the cases which I have seen in which it has been done does not give a satisfactory result, and this is what one would expect from the anatomy of prolapse, as it simply leaves the hernia, minus the uterus, which results in a great bulge of the vaginal vault full of intestine, and causing as much discomfort as the original trouble.

In conclusion, there are certain cases which from accompanying circumstances will have to be treated by pessaries, and in these cases it is important that the patient realises the necessity of a regular visit to the doctor, if they are unable to replace the pessary themselves. If they are I make them take it out at frequent intervals and use regular douches; if they are not, to report themselves at intervals of about eight weeks, so that the pessary can be seen to.

I have seen both the rubber pessary rusted through and a foreign make of Hodge perished and ulcerating through the vaginal walls, both due to neglect of regular medical attendance.

If patients, however, can be operated on, there are several methods by which their ailment can be greatly alleviated, and just as in the case of radical cure for hernia, which is nowadays performed so frequently and satisfactorily, a woman who is subject of hernia in another form ought to be given the chance of being relieved from all the discomforts and inconveniences of pessary treatment.

August 2nd, 1915.

Dislocation of the Semilunar Bone.—Runyan says that careful examination of all carpal injuries and skiagrams are necessary for diagnosis. Reduction can be accomplished in about one half of the recent cases by hyperextension followed by flexion while counter-pressure is maintained over the dislocated semilunar. Prompt diagnosis and treatment are essential in obtaining good results. The more speedily the dislocation is recognised and reduced the better the result. Closed reduction should always be tried, irrespective of the duration of the dislocation. Closed reduction being impossible, an anterior incision should be made and the dislocation reduced. Failing this, it is necessary to excise the semilunar as a last resort.—*American Journal of Med. Sciences*, p. 763.

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We regret that owing to the war the number of pages must be diminished for a time.

THE OPERATIVE TREATMENT OF CERTAIN FRACTURES OF THE LOWER EXTREMITIES IN CHILDREN.

By JOHN H. WATSON, M.B., B.S., F.R.C.S.,
Surgeon, Victoria Hospital, Burnley.

DURING the last eight years it has been my lot to treat a considerable number of children who have sustained fractures involving one or other of the long bones of the lower extremity. Until the year 1914 I had been content to use the so-called "closed method," trying many and various forms of retentive apparatus according to circumstances. The results, I may say, have also been variable. At times I have been elated by obtaining excellent union in every respect. More often I have had to be satisfied with what is usually designated a moderate result, which really means a useful limb with some slight deformity, and occasionally with a really bad union giving rise to such marked deformity, shortening and disability as to make one think that the method employed has been inadequate or carelessly and imperfectly carried out.

This last class of results, although rare, seem peculiar to small boys, and are enough to make one reconsider the plan of treatment. These are restless, mischievous urchins, especially difficult to deal with even in hospital. They wriggle about in bed and displace their splints and traction apparatus, although most carefully watched. The nature of the fracture is invariably of the spiral or oblique type, and is caused by some form of indirect violence sustained in falling from walls, trees, or the backs of carriages, or other vehicles. I have found them difficult to reduce by the closed method and, even if reduced satisfactorily, extremely liable to displacement after the most careful splinting. These cases demand more than the average all-round care and skill, so essential to the treatment of fractures, and nothing is more fallacious than to

assume that fractures in children are simpler and easier to treat than those of adults. I believe to properly diagnose, reduce and care for a fractured limb requires from the surgeon more experience and skill than is ordinarily displayed in undertaking an operation for gall-stones or appendicitis.

To the layman nowadays a malunited fracture means poor surgery, and may obtain for an unfortunate practitioner a bad reputation and a most undesirable publicity in the law courts. More especially it behoves us to be extremely careful to obtain good results, when one's work is shown up so vividly by X-ray photographs. If, therefore, we undertake to treat fractures, we should base our treatment on certain principles. Rutherford Morison's (1) rules are trite and to the point, namely:

(1) Apply first aid.

(2) Examine the complications, local and general.

(3) Reduce deformity.

(4) Keep it reduced.

(5) Restore function.

I would, however, insist that before attempting to reduce the fracture an X-ray photograph should be obtained if possible, and a second taken at an early date to verify the correction or otherwise show the remaining displacement.

These rules are all right so far as they go, but we may well ask ourselves, as Estes (2) suggests:

(1) What is the best treatment for the particular fracture with regard to (*a*) the nature of the fracture; (*b*) the general condition of the patient; (*c*) the environment?

(2) What method will give the patient the least suffering and discomfort after reduction and fixation? and

(3) Which method is likely to result in the shortest disability, and give afterwards the maximum functional result?

(1) *What is the best treatment for the particular fracture, etc.?*—The particular fractures to which I wish to draw attention in this paper are the oblique and spiral fractures of the femur and tibia. These fractures I maintain are not easily reduced, and even if we are successful in getting a good anatomical alignment, displacements very often recur after applying what is thought to be a suitable retentive apparatus. In the majority of these cases I doubt the possibility of obtaining anything like a good anatomical apposition of the fragments, owing to the intervention of muscle, periosteal flaps or splintered bone which I have frequently observed in the way at an open reduction.

The evil results of an imperfect reduction by the closed method, apart from the discomfort to the patient, are an endless cause of anxiety to the surgeon. A delayed union, over-riding, or axial rotation of the lower fragment are enough to contend with, but to be presented with a radiograph showing an angular deformity is, to say the least, disconcerting. In these cases there is invariably a considerable stripping and laceration of the periosteum, so that osteoblasts are scattered indiscriminately in the surrounding tissues, thus producing an irregular and exuberant callus which intensifies the deformity.

Lane (3) states in his book that "If in the young child a long bone is broken and the fragments are displaced, a portion of the original shaft is removed and a new one transmitting force from the two articular extremities of the bones is laid down," and he demonstrates his statement by a beautiful series of radiographs, showing the process in the fractured humerus of a child. In the lower extremity there will be a certain deflection of the weight-bearing axis of the limb, which gives rise sooner or later to more or less disabling changes in the joints of the extremity, so that the deformity has far-reaching effects, although, as he says, Nature endeavours to restore as far as possible the normal form.

I have now no hesitation in saying that we should adopt Lane's methods in this particular type of fractured lower extremity, for, to the patient, the ultimate benefit is incalculable.

If, therefore, we are not capable of carrying it out ourselves, by all means transfer the case to someone who can, for I do not believe that accurate adjustment of the fragments is practically ever obtained by the old conservative measures.

(2) *What method will give the patient the least suffering and discomfort after reduction and fixation?*—My own experience with these cases has fully shown to me that after the reaction has passed over from the operation the patients give very little trouble and rarely complain of any further pain or discomfort. This is in absolute accord with the observations of the house surgeons and ward sisters.

(3) *Which method is likely to result in the shortest disability, and give afterwards the maximum functional result?*—There is not a great deal to choose between the operative and conservative methods as to the period of disability, but I am firmly convinced that the metal plate does not delay union if properly applied. I have only had one experience of delayed union, and that was in a compound fracture in which a sharp fragment of bone had punctured the skin. Some fourteen days later, under the impression that the wound was absolutely innocuous, we operated, and had the mortification of finding pus oozing from the site of the original wound after a few days had elapsed, so that the plate had eventually to come out, but the bones remained in good position and healed well.

The remarkable statement that the plate inhibits osteogenesis is, I believe, an American observation. I have myself no doubt that with a successful plating the best functional result in every respect is obtained.

At this point I would like to refer to the findings of the Committee appointed by the British Medical Association to consider the question of the treatment of fractures, which may be summarised thus:

(1) The best way to obtain a good functional result is to secure anatomical replacement, but it is undoubted that a useful limb may be obtained with an indifferent anatomical correction.

(2) In practically all age-groups operation cases show a higher percentage of good results than the non-operative cases.

(3) No method, non-operative or operative, which does not promise a good anatomical result should be accepted as the method of choice.

From these conclusions, drawn up by a representative body of British surgeons, we have, therefore, every encouragement to adopt operative treatment in suitable cases in children, granted we have all essentials for such treatment. The actual figures given in the cases of children under fifteen are 1016 cases treated by conservative measures, of which 90 per cent. obtained a good functional result, whereas only 64 cases were recorded which had submitted to operation with a satisfactory result in 93 per cent. However, an improvement of 3 per cent. is certainly worth trying for, but in my experience it is much greater than this.

I cannot acquiesce in the declaration that fractures in children never require operation. In most cases they can undoubtedly be reduced and maintained in position by non-operative means, but these are usually fractures with comparatively slight displacement—the so-called "parosteal fractures"—in which the periosteum is practically intact. If we recall the structure of these young bones, one of the striking features is the relatively thick and strong periosteal layer with its intensely active osteogenetic cells, capable of throwing out callus rapidly and hence accomplishing repairs more quickly. Added to this the bones are more flexible, and we get in children the peculiar type known as "green-stick fracture." These and other transverse fractures do perfectly well with ordinary conservative measures.

The fractures of the lower extremity, which I would submit to operative measures, other conditions being favourable, are:

(1) Certain spiral or oblique fractures of the femur.

(2) Fractures with interposed soft parts, *e. g.*, muscle, fasciæ.

(3) Certain oblique fractures of both bones of the leg.

(4) Certain fractures near the knee and ankle joints.

(*N.B.*—It must be remembered that we are dealing with growing bones, and if a plate is inserted across a growing epiphysial line it will require removal at an early date.)

These, for the most part, so far as my experience goes, do not give such good results with the conservative measures as are to be obtained by the modern operative treatment.

As an illustration I would quote the case of H. S—, æt. 6 years, who, on March 5th, was playing in the school-ground when he slipped on a piece of orange peel and fractured his left thigh. He was admitted to hospital on March 7th; on March 9th a radiogram was taken (Fig. 1). A day later he began with an attack of tonsillitis and ran a temperature for fourteen days. During this period conservative measures were adopted to straighten the thigh by means of a Bucks extension and a long outside splint. On March 30th a second radiogram was obtained (Fig. 2) showing an angular deformity with an excessive deposit of callus, which could easily be felt as a hard ridge. An opera-

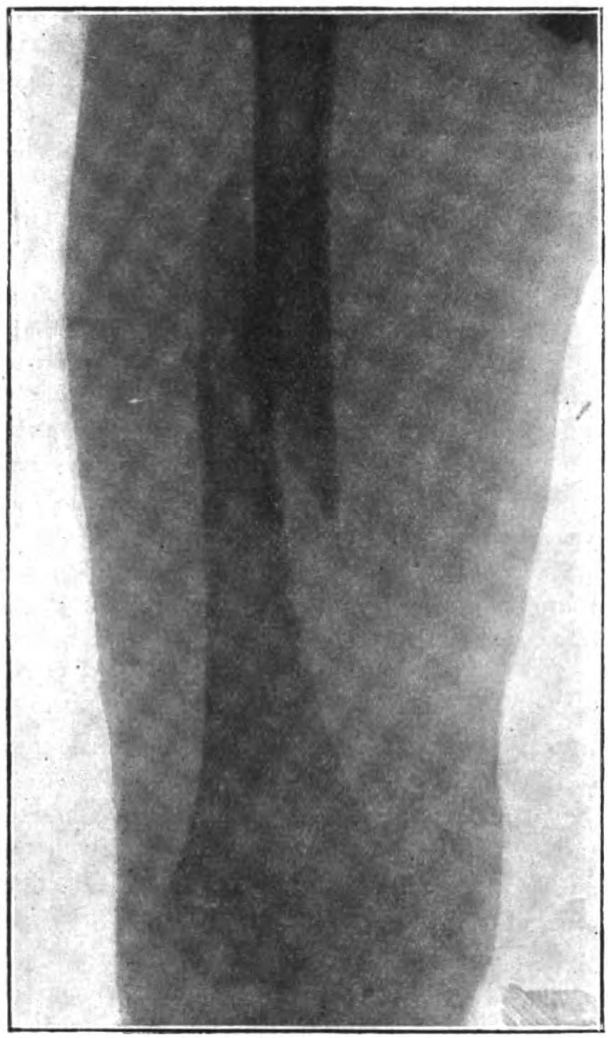

FIG. 1.—Skiagram, showing spiral fracture of femur, two days after injury.

tion was performed on April 7th. The exuberant callus was removed. A flap of periosteum was found interposed between the fragments, the sharp points of which were impaled in the surrounding muscles. The fragments were eventually placed in exact position and plated. On May 4th a third radiogram was taken, four weeks after the operation, showing an excellent correction (Fig. 3).

The advantages to be gained by this open treatment are so admirably given by Lane (4) himself that I have taken the liberty to quote him here in full.

"(*a*) They at once relieve the patient from the pain produced by any movement of the fragments upon one another.

"(*b*) They free him from the tension and discomfort due to the extensive extravasation of blood between and into the tissues.

"(*c*) They shorten the duration of the peri d during which he is incapacitated from work, since union is practically by first intention and consequently very rapid and perfect.

"(*d*) Lastly, and by far the most important, they leave his skeletal mechanics in the condition in which they were before he sustained the injury."

These findings apply in every instance to the operative treatment in children. Sampson, in a

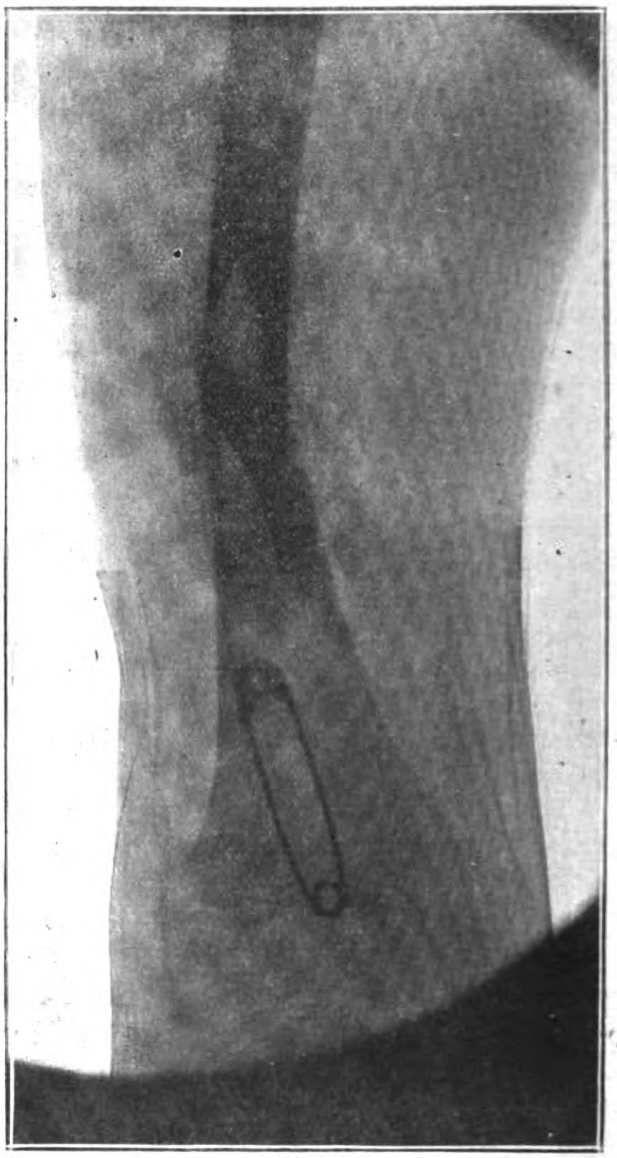

FIG. 2.—Skiagram, taken three weeks later, showing deformity of the femur and exuberant callus, notwithstanding use of splints and an extension.

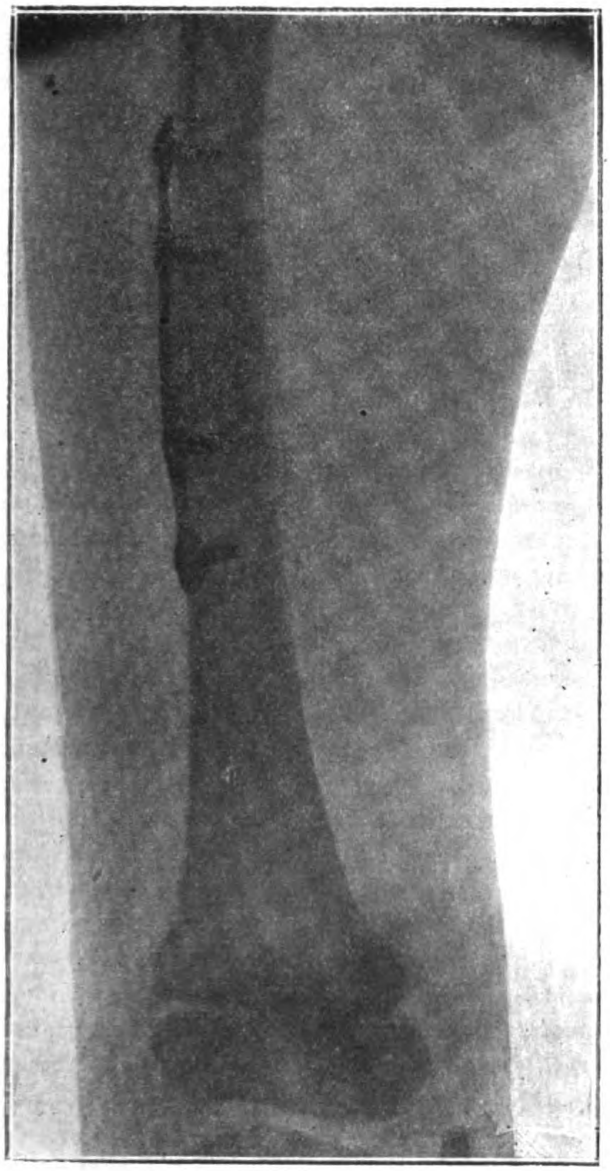

FIG. 3.—Skiagram, showing condition of femur four weeks after operation.

paper to the 'Lancet,' August 17th, 1912, gives his views on the operative treatment of simple fractures of the long bones in children, which are most instructive and may be summed up as follows:

(1) The treatment of simple fractures by operation gives more perfect results than can be obtained by any other method at present in general use.

(2) With reasonable care the dangers of such treatment are negligible, the mortality has been *nil*, and no instance of wound infection has occurred in a complete series of cases.

(3) The insertion of a metal plate gives no trouble after a clean operation.

(4) Operative treatment should be applied to recent fractures and not reserved for the imperfect results of conservative measures. The chance of a perfect result being obtained is diminished by the length of the period which elapses between the accident and the operation.

(5) Rarefying osteitis and sinus formation do not occur after a clean operation.

(6) In view of these results it is reasonable to urge the more extended adoption of Lane's methods for the treatment of simple fractures.

These deductions call for criticism, and first I would put special emphasis on the phrase "with reasonable care." I have seen many operations on bones—good, bad and indifferent—but it has also been my good fortune to watch Sir Arbuthnot Lane operate on these fractures on several occasions and to observe his careful technique, upon which alone success is to be founded and ensured. The technique needs to be seen to be appreciated. Practically it means an operation conducted with inviolable aseptic detail by a most skilful and careful operator, using a special armamentarium which he has himself devised, and assisted by a specially trained staff. Reasonable care means the absolute adoption of this technique. The absolute contra-indications are an indifferent surgeon who cannot keep his fingers out of the wound and has not a thorough grasp of the anatomy of the part, untrained assistants, unsuitable environment, and an incomplete equipment.

Again, not only must the operation be a clean one, but it is essential that there be no infected wounds or abrasions near the site of the operation and that the general condition of the patient be good.

Granted, therefore, that we have a straightforward case upon which to operate, I will, as shortly as possible, outline the steps of the operation.

(1) Special care is to be taken in the preparation of the skin in the neighbourhood of the fracture. As a rule, after most thorough cleaning with soap and water and spirit, the iodine method is adopted. If the skin is coarse, I have used a cream made of equal parts of chlorinated lime and carbonate of soda, which is stirred up with a little water and then swabbed over the part for a few minutes. The nascent chlorine is a very powerful germicide,

and the alkali gets rid of the superficial epithelium. After this preliminary step the cream is rapidly washed off with sterile water and spirit, for, if left too long, it is a powerful irritant, but it is a useful adjuvant, permitting deeper penetration of the iodine.

(2) It is my practice to mark intervals of about one inch across the line of the proposed incision with a needle. These scratches serve as landmarks later for the accurate apposition of the edges of the wound.

(3) The incision is now made which should be large enough to allow easy working. After incising the skin it is excluded at once by attaching sterile towels or, better still, thin rubber sheeting covered with gauze, which are held in position with tenaculum forceps.

(4) The wound is deepened down to the bone and any blood-clots turned out. The periosteum is incised and the fragments freed with the aid of one or more special elevators. The entire operation is done with instruments. Even the operator's gloved hand is not permitted to come into contact with any portion of the wound. The instruments are best brought in a container direct from the steriliser, and are not permitted to come into contact with any other object, and are used directly from this tray. Swabs are taken out of bundles recently sterilised with long forceps, and are held in the forceps to dry the wound.

(5) The periosteum is preferably stripped from the bone with the other soft parts with care. It is an added and unnecessary labour to separate it from the overlying muscles. Therefore, if possible in the first place, make a firm incision through muscle and periosteum on to the bone; the plate is applied under the periosteum.

(6) After freeing the fragments be careful to scoop out blood-clots and partially organised *débris* from the broken ends, and thus freshen them.

(7) Hæmorrhage is controlled by grasping any bleeding points with strong hæmostatic forceps (Mayo Ochsner's), and these are left on until the completion of the operation. When removed it is very seldom that there is any need to use a ligature. Only rarely have I found it necessary to use one or two catgut ligatures.

(8) Accurate anatomical alignment must be secured, and this is accomplished by grasping and levering the ends of each fragment into apposition with Lane's bone-holding forceps.

(9) The size of the plate should be selected according to the strain likely to be placed upon it. Personally, I prefer the thin vanadium steel plates of Sherman, using one or more if necessary.

(10) In drilling holes for the reception of the screws, be particular that the bore is less than that of the screw, so that the latter will fit tightly. The screws also should be long enough to comfortably penetrate the layer of the compact bone.

(11) The wound is closed after being swabbed quite dry. The muscles and other tissues fall into place without suturing, and the edges of the skin are brought accurately together with Michel's clips. I prefer to use three or four relaxation sutures of stout silkworm gut introduced from within out, taking up muscle fascia and skin at points already marked on the surface. These are knotted at the stitch holes, not over the line of incision. The margins of the wound are then closely approximated by a fine silkworm suture taking up the skin only.

(12) A sterile dressing is applied and the limb immobilised. I believe it to be of no little importance to keep the limb absolutely still until the stitches are removed on the tenth day, and a few days longer in some cases. In order to do this in the case of the thigh, the limb is placed in the most restful position, that is with the knee and hip partially flexed, and it is retained in this position by a specially moulded plaster splint (the drawings show exactly the form adopted). A gutter splint is made to embrace half the circumference of the limb extending from just below the buttock down the back of the thigh and legs under the heel to the middle of the sole. The shape is made from house flannel and is slit at the knee and heel sufficiently to allow proper moulding. Three layers are taken and soaked in thin plaster and then moulded to the limb after suitable padding. The edges then are hemmed with adhesive plaster (*vide* Fig. 4). For the lower limb two lateral splints, after the fashion of Croft splints, are made of three layers of coarse house flannel soaked in thin plaster. The pattern for the inner side of the leg should take in the foot and reach just below the knee. The shape for the outer side extends from the ankle to just above the knee (Fig. 5). When both splints are applied after suitable padding, the knee and ankle-joints are both firmly fixed, whereas if one or other

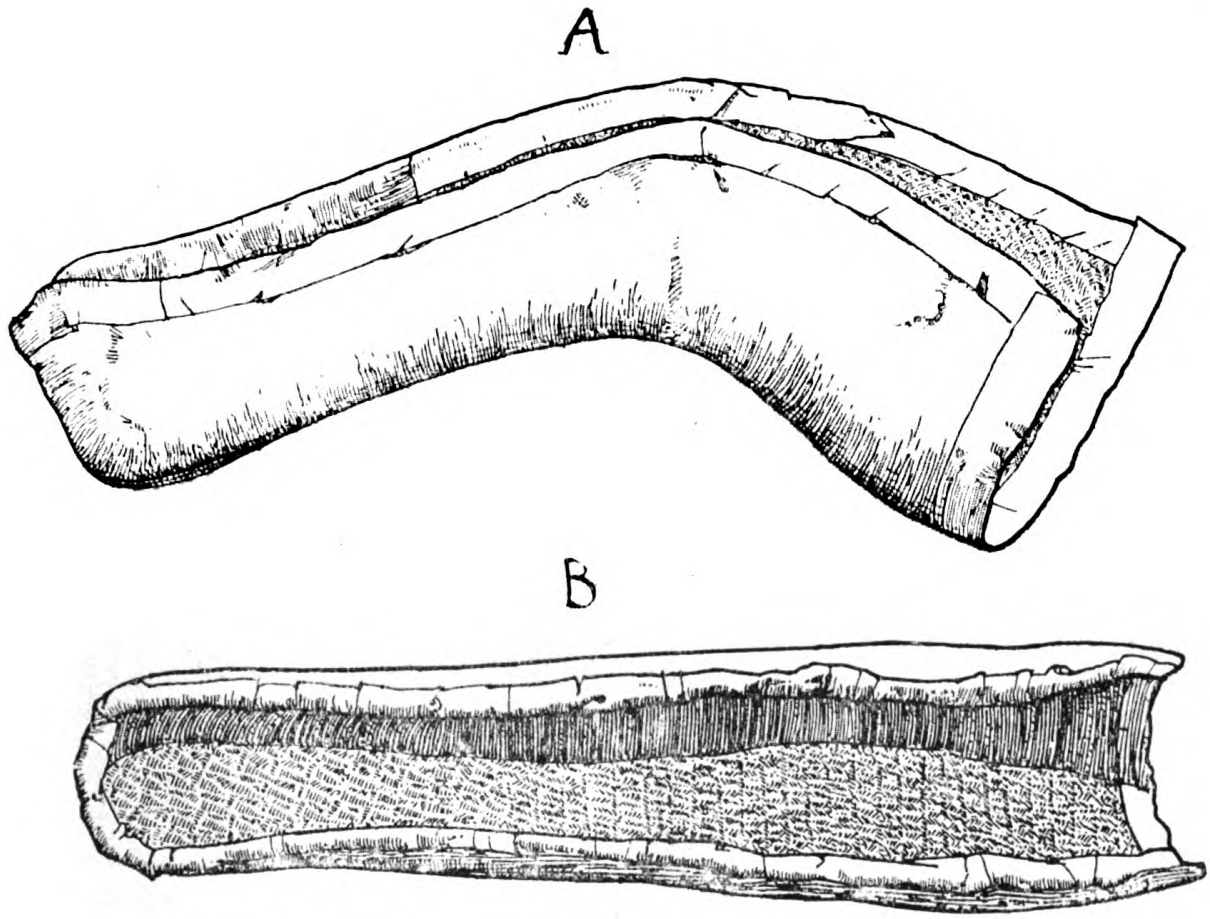

FIG. 4.—Plaster splint for encasing lower limb after operative treatment of fractured thigh. A. Lateral aspect. B. Viewed from above.

is removed passive movements may be employed either at the ankle or knee joint I have found these the most serviceable and economical type of splint, as they ensure absolute immobility, and the child cannot very well displace them. The splints are prepared beforehand so that they are ready for immediate application at the end of the operation.

Opinion is divided on the question of the most suitable time for operation. Some advocate operating in the first twenty-four hours. Others advise a delay up to seven to fourteen days, for they say that by this time the exudate in the region of the fracture has been absorbed and reaction in the tissues about the injured areas has set in, whereas that in the first twenty-four hours they hold that the patient is suffering from shock, that the tissues are infiltrated with blood and more liable to infection, that the skin is bruised and œdematous, and therefore it is more difficult to ensure surgical cleanliness, and so on, according to the view the surgeon particularly wishes to uphold. What does matter, however, from the operating surgeon's point of view is to find the fragments in a mass of callus of varying consistency, needing removal and enhancing the difficulties of obtaining a good position, and judging from the pathology of bone regeneration we must remember that osteoid deposits are beginning to be laid down about the twelfth day. It may therefore be concluded that operative procedures should be undertaken if possible before that time, and, inasmuch as it is unproven that the results are better after an immediate operation than after one delayed ten days, it may be accepted that any time within the first ten days is suitable.

Before concluding I cannot refrain from quoting a surgeon who wrote in THE CLINICAL JOURNAL

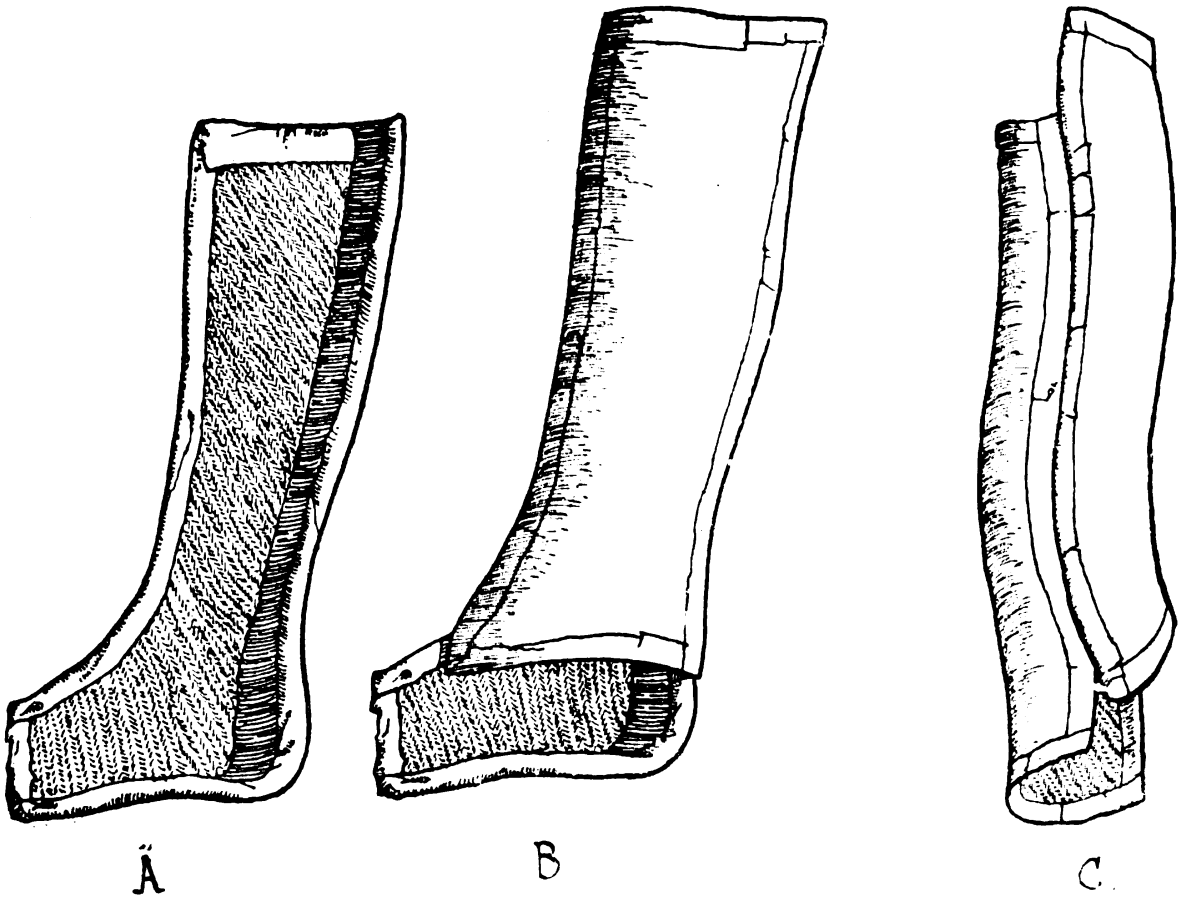

FIG. 5.—Plaster splints used after operation for immobilisation of the leg. A. Inner splint from inner aspect. B. Both splints in position, from outer aspect. C. Both splints from the front.

twenty years ago with regard to Lane's work, and in which he refers to the "the folly of attempting at once to fix a bad fracture in accurate position." In the light of present-day surgery this was a most unfortunate and fallacious remark. It is, however, a good indication of what Lane had to contend with in his earlier work. Acute disputations still continue on the merits and demerits of the Lane plate. Other complicated treatments are advanced such as the various elaborate and costly forms of traction apparatus, the semi-operative traction methods of Steinman, the external clamps and screws of Lambotte, the inlaid bone-graft of Albee, but for my own part I am thoroughly convinced of the efficiency of the Lane plate, provided it is used with proper precautions and reasonable skill. As I have implied before, it is not meant for the rough and ready surgeon or the occasional operator. Lane's work marks a new epoch in the surgery of fractures. It is beyond all praise, and it is gratifying to his followers to know that his indomitable perseverance in this pioneer work has at last commanded universal attention and recognition.

I should like to acknowledge my indebtedness to Dr. Crump for the skiagrams and to Mr. Noel Leaver for the care he has taken in sketching the splints.

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August 9th, 1915.

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We regret that owing to the war the number of pages must be diminished for a time.

FOUR GRESHAM LECTURES ON HEREDITY.

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

LECTURE I.

The Science of Heredity.

In his 'Descent of Man,' Charles Darwin writes: "Man scans with scrupulous care the character and pedigree of his horses, cattle and dogs before he matches them; but when he comes to his own marriage he rarely, or never, takes any such care. He is impelled by nearly the same motives as the lower animals, when they are left to their own free choice; though he is so far superior to them that he highly values mental charms and virtues. On the other hand, he is strongly attracted by mere wealth or rank. Yet he might by selection do something, not only for the bodily constitution and frame of his offspring, but for their intellectual and moral qualities. Both sexes ought to refrain from marriage, if they are in any marked degree inferior in body or mind; but such hopes are Utopian and will never be even partially realised until the laws of inheritance are thoroughly known. Everyone does good service who aids towards this end."

Why, we may ask, has the study of the laws of inheritance been so neglected? Why has man given so little thought to what concerns him so very nearly? There are, of course, several answers to this question. Some have trusted that instinct and their natural passions would prove the wisest guides, others have honestly believed that good nurture and healthy surroundings would obviate the faults of bad breeding; the vast majority of mankind has acted on blind impulse only and has not given the matter a thought.

"Marriages are made in Heaven" is an old saying, and I am not one to deny that the best of them are above all earthly criticism. But it would

require a very wide stretch of imagination to think that some others proceed from anything higher than very elementary needs, in spite of the fact that most marriages are entered upon in some form of love. Mr. Paterson, in his admirable description of South London life among the riverside population, says:

"At Christmas and Easter boys and girls are married, not in couples, but in batches. The service is cut short, but not made thereby any easier to understand. It is hurried through on both sides; the honeymoon lasts but for a few hours, and is spent inside a public-house near the church. The chance worshipper in the church would realise with horror that this was the crisis in two lives, and the signal for the birth of many more."

A wise statesman, putting the question: "What is the greatest thing in the world?" gave the answer himself: "A baby!" For the baby of to-day is the citizen of to-morrow, who will be the strength or the weakness of England in the days that are to come.

Two thousand years ago Plato, the Athenian philosopher, foreshadowed much that the world has thought good to strive for many centuries later. Jowett has said of him that "he is the father of idealism, in philosophy, in politics, in literature. And many of the latest conceptions of modern thinkers and statesmen, such as the unity of knowledge, the reign of law, and the equality of the sexes, have been anticipated in a dream by him." In the 'Republic' he expresses his ideals of government, in a discussion, which is supposed to be carried on between Socrates and his friends; and no more ardent advocate of race improvement could be found. Socrates points out with what care the best dogs, birds, horses, and other animals are mated by man in order to produce the best young; and he suggests that the union of citizens should receive as great attention. The best man, especially he who has distinguished himself by his valour, should have more wives than others. "He is to have first choice in such matters more than others, in order that he may have as many children as possible." Great hymeneal festivals should be inaugurated; and the best must marry the best, the worst the worst. The children of the best should be carefully nurtured in public crêches, the children of the worst should be put away in "unknown places." "People must be acquainted with those into whose family they marry, and to whom they are given in marriage."

His schemes were not very practical, and they were probably not meant to be accepted literally; but the basis of his teaching is sound; it was obvious to him, as it is to thinking men and women now, that a policy of leaving well (or ill) alone has not worked to the best possible advantage of the race, and that to mend matters the nettle must be grasped, and we must understand clearly where we stand and to what dangers we are heedlessly drifting.

The more thoughtful man or woman, whose life is guided by a desire to make this world a little bit better for his or her presence in it, and whose sense of responsibility has been awakened regarding the question of heredity, which involves the future of the race, finds himself surrounded with much prejudice and many superstitions. But although the subject is still bristling with difficulties, and there remains far more to find out than is already understood, certain facts are known and established beyond doubt, and here, as in all natural science, great strides have been made in the last decade.

It is known and has become a commonplace, though marvellous, biological fact, that the individual life of almost all animals and plants originates in the fusion of two infinitely small elements, the male and the female germ cells, uniting to form a new being, which possesses the attributes of the parents conveyed through the union of the two cells. Thus the material basis of inheritance is dual and lies in the fertilised egg cell. We need not enter into the few exceptional cases of asexual multiplication, which takes place in certain low forms of animal life.

"The entire organism," Huxley maintained in 1878, "may be compared to a web of which the warp is derived from the female and the woof from the male." This exact division of paternal and maternal contributions has been confirmed by recent discoveries in embryology.

Heredity is described as "the organic or genetic relation between successive generations" and inheritance as "all that the organism is, or has to start with, in virtue of its hereditary relation to parents and ancestors."

Each generation resembles its ancestry in all its wonderful complexity, and this fact we accept

naturally and as a matter of course. It is only when the offspring develops some special bodily or mental attribute characteristic of a member or members of his ancestry, that we are struck with the fact and speak of his inheritance. It is such special characteristics, good or bad, which make it obvious that the study of heredity is one of vast and far-reaching importance.

The question has been attacked by many authorities, from very early days and in many ways, and we may divide the more scientific methods into three groups: the microscopical, the experimental, and the statistical; through each of which we have gained substantial knowledge.

What may be termed the microscopical method, because it deals with the infinitely small germinal matter from which the complex organism springs, has found its strongest exponent in Prof. August Weismann, of Freiburg, to whose genius we owe the theory of germinal continuity, propounded by him some thirty years ago, a theory now almost universally accepted, which explains many observations for which no adequate solution had previously been found.

Very briefly the theory is this: In the formation of a new individual it is the union of germ plasm, contributed by each parent, which is the material from which it springs, and, whilst some of this small mass of germ plasm goes to build up the new body, another portion is set aside within the body of the growing individual and remains unchanged for the formation of the following generation. "The germ cells, therefore, contain within them two constituents, the somatic or body plasm and the germ plasm."

Thus each generation is literally linked with the last in a continuous chain, and the likeness expressed in the individual, not only to his parents, but to former generations, becomes easy to understand. In the words of Prof. Karl Pearson: "The bodily resemblance of parent and offspring arises . . . because both are the product, in the moiety, of a continuous germ plasm."

The fact that each individual is, however, the result of a duplicate ancestry, paternal and maternal, complicates such a likeness and is the chief cause of variation in the type.

In the building up of each individual there lie the contributions, bodily and mental, of his ancestry, any one of which may, on the one hand, find expression through the influence of nurture, or through a given stimulus, or through the power of a duplicate inheritance, that is to say, where it has been inherited from both sides of the family. On the other hand, any one characteristic may never assert itself, but may remain as a potential peculiarity only, repressed by nurture, unable to express itself through lack of stimulus, or counterbalanced by a dominating inheritance from the other lineage. The dissimilarity of an offspring to its ancestry is, however, less often real than apparent, and when any resemblance reappears in the next generation it proves that it was present, though not expressed.

Weismann has elaborated another theory in answer to the question why some physical and mental features persist in families whilst others disappear; why each individual is not an exact mosaic of ancestral contributions. He accounts for this by the ingenious hypothesis that a struggle for expression takes place among the hereditary tendencies, which determines the predominance of certain characteristics as well as their extinction. This theory of germinal selection also accounts for the disappearance of useless parts of the animal organism, a fact which has never been satisfactorily explained by natural selection only.

Weismann considers that the germ plasm is composed of "an army of living determinants," the potential parts of the organism. Suppose the determinant of any given part not to be present in the germ plasm, that part will be absent in the full-grown organism; and this is not a matter of speculation, for it has been demonstrated by experiment in some low forms of animal life. These determinants have the power of feeding, growing and multiplying, and the more vigorous determinants will assimilate more of the nutrient matter supplied to the germ cell, whilst the weaker ones get less. As soon as any determinant is superseded by others and is less able to struggle for its food supply, it begins its downward path and dwindles. If the structure of which it is the determinant is useful to the individual, it will again assert itself through natural selection, those individuals possessing it having an advantage over those who have not, and therefore being more successful in reproducing their kind. If the structure is not useful, natural selection does not operate, and the limit of dwindling is only reached with its ultimate extinction.

As an illustration we are given the case of the

whale, whose hind legs can still be seen in the bony structure of the skeleton, although they have in the course of evolution disappeared below the surface of the skin. Natural selection must first have operated in favouring those whales which, using their tails for swimming, were not impeded by the presence of large hind legs. The whales with lesser legs, therefore, having the advantage in moving quicker to find food and their mates, were the individuals whose hind leg determinants were less vigorous than certain others. These determinants, being less vigorous, were on the downward grade, and the hind legs continued during many centuries to dwindle, even when they had reached the level of being below the skin and therefore being no longer an obstacle to the swimming of the whale.

The theory explains not only the disappearance of useless parts, but the increase of useful structures, those individuals being favoured in the struggle for existence whose determinants of those parts were on the upward grade. Stronger determinants tend to become stronger by their power of assimilation; weaker determinants tend to become still weaker by their lessened power of assimilation, and thus "the germinal selection fosters and strengthens personal selection."

If we accept, as we are bound to do by the continued investigation of biologists, Weismann's theory of germinal continuity, we must believe the germ plasm, from which the next generation springs, to be something relatively apart from the body, segregated at a very early stage in the development of the parent body in which it lives, and not easily or rapidly influenced by external impressions; but we must not fall into the serious error, which has unfortunately become a common one, of assuming that what happens to the parent body can never influence these reproductive cells. None who follow Weismann's teaching carefully can believe this. It must be remembered that the germ plasm is nourished and harboured in the body, and any prolonged debilitating change in the body must diminish the vitality of the germ cells, whilst any increase in vigour of the body must increase their vitality.

I shall return to this subject later, as it is of paramount importance in dealing with the practical side of heredity.

The masterly work of Weismann has commanded the respect and attention even of those who do not altogether accept his views. He has, in an eminent degree, the power of drawing brilliant conclusions from what his microscope reveals, and building these up by the scope of an imagination kept within bounds by his scientific rectitude, a power which distinguishes the man of genius from the mere working scientist.

Whilst Weismann is the greatest microscopical exponent of heredity, Mendel has enlarged our knowledge by the experimental method. Experiments in heredity can obviously only be carried out in plants and animals. It is from the results of breeders that we have drawn many of our conclusions; but, very suggestive as their results are, they are often complicated by old-time superstitions which die hard, and by the lack of scientific training in the experimenters, a lack which leads to inaccuracy and a disregard for detail which often makes their results inconclusive and worthless.

Gregor Johamm Mendel was born in Austrian Silesia, of good peasant stock, in the year 1822. He entered the Monastery of Altbrunn at the age of twenty-one, but left it in 1851 to study physics and natural science for two years in Vienna. He returned to his monastery, became a teacher, and ultimately abbot.

During his leisure hours, it was his pleasure to work out experiments in the hybridisation of peas and other plants in the quiet garden of the monastery; and though he published two papers, one dealing with his experiments on peas with his conclusions, and a shorter one on hawkweeds, it is not likely that he himself realised, any more than the few who read them, the far-reaching importance of his discoveries.

For many years his paper on peas, dealing with the inbreeding of hybrids, lay buried in the 'Proceedings of the Natural History Society of Brunn.' In 1884 the worthy abbot died.

In 1900, Mendel's law, as his conclusions have come to be called, was re-discovered by botanists; and since then others, notably Prof. William Bateson, F.R.S., have worked out, confirmed and extended his experiments, and they are now almost universally accepted.

He selected for his experiments the edible pea, because different varieties have very distinctive characters; because it is habitually self-fertilised: and because it is also easily artificially fertilised.

His experiments were made to discover the laws which govern the inheritance of qualities in hybrids. Selecting peas of different varieties, such as tall and dwarf, or with wrinkled seeds and smooth seeds, or with different coloured seed coats, he proceeded to make crosses between them, studying one type at a time, by taking the pollen of one variety to fertilise the stigma of another variety.

The results proved to be surprisingly regular. When a tall variety was crossed with a short, all the next generation was tall; there were never any intermediate forms; and Mendel called the tallness the dominant, the shortness the recessive quality. When a yellow-seeded pea was crossed with a green-seeded pea all the offsprings of the first generation were yellow; yellow being dominant, green recessive. In crossing two varieties, therefore, the first generation shows only the dominant type, the recessive is apparently suppressed. But, when the cross bred plants were allowed to fertilise themselves, the two original forms reappeared in the next generation, tall and short, or green and yellow, on an average in the proportion of three dominant to one recessive. The recessive, again self-fertilised, continued to breed true, producing recessive only, but the dominant, self-fertilised, bred one third pure dominant, which continued to breed true, and two thirds cross bred dominants, which gave rise in the next generation to dominants and recessives again in the proportion three to one.

This will be easier understood by looking at a diagram. Mendel gave a theoretical interpretation to these facts, which it is difficult to condense as I should wish. He affirmed that the inheritance was built up of gametes, or items, any one of which might be the dominant or recessive type, items which correspond to Weismann's determinants. The parent cannot pass on to his offspring an element which he does not possess himself. The offspring formed from the union of two organisms, both being pure bred in respect of certain characters for good or ill, must possess those characters and must be free from all taint of the cross.

This applies to any character in botany, zoology, and among humans.

Experiments to confirm Mendel's observations have been carried out on peas, maize, and other plants, in mice, rabbits, and poultry, as regards colour and some other peculiarities, and the truth of the law has been established in a large number of dissimilar characters, though not in all. The application of his laws must be of great value to the stock breeder and to the gardener, who can attain their ends more surely and with fewer disappointments if they follow his rules. Do these laws apply to man? Undoubtedly it will be found increasingly that they do, and the understanding of these laws will make it clear that the marriage of good and bad stock will not, in spite of good nurture and outside influence, necessarily eradicate the bad strain.

Where a parent has a neurotic or unsound heredity, the immediate offspring may not show that strain, but it may be lying latent to reappear in the next generation, though by fortunate mating such a character may in some cases altogether disappear.

"Definite characters for good or evil," says Prof. J. A. Thomson, "do not disappear in Mendelian inheritance." They persistently reappear in their original "purity"; and Prof. Punnett writes: "Permanent progress is a question of breeding rather than of pedagogics, a matter of gametes, not of training. As our knowledge of heredity clears, and the mists of superstition are dispelled, there grows upon us with an ever-increasing and relentless force the conviction that the creature is not made but born."

It is very difficult to study Mendelian inheritance in man, because few men can be sure of a pure strain in any given characteristic; it would need an accurate knowledge of his ancestry, which it is hardly possible to get, and having got it we should generally find that he was hybrid in almost any direction. But cases are to be found of the intermarriage of black and white people, and the pedigrees of royal families (which are well known and often consanguineous) might be obtained; and from both these sources the law might be studied, but the generations of men follow each other so slowly, and the individuals are comparatively speaking so few, that we must admit that man does not lend himself easily to the study from the experimental point of view, although to most of us the interest in the question centres around him.

Where the question has been studied in man, it is chiefly in unimportant trifles, in diseases and in abnormalities. It has been found to be well illustrated in man in eye colour. We may divide

human eyes into brown and blue, for blue or grey eyes have no pigment in front but only a layer at the back of the iris, and they appear blue or grey according to the transparency of the iris itself. Brown eyes, which include hazel, green or black, have a layer of pigment in front of the iris, varying in quantity, which gives the variety in colour. Brown eyes appear to be dominant, so that if a pure bred brown-eyed person marries a blue-eyed individual, all the children are brown-eyed (with the blueness lying latent). If any of these hybrid brown-eyed children marries a blue-eyed person or another hybrid brown-eyed like himself, they will have both brown- and blue-eyed children. But two blue-eyed parents (this being pure recessive) can only have blue-eyed children.

Mendelian inheritance is all too plainly apparent in certain deformities of which I must speak later, and their occurrence in the offspring may be foretold with painful certainty, so that in this respect also there is practical utility in the understanding of the laws of inheritance.

Prof. Bateson, Mendel's most enthusiastic exponent, maintains that "no one before the rediscovery of Mendel's work had ventured to imagine that the confusion, the paradoxes, the capricious disorder of the phenomena of descent were, in very great measure, capable of a simple and ready analysis."

Before proceeding to the third method of studying heredity, the statistical method, of which the late Sir Francis Galton was the chief and most successful advocate, it is well to apply what we learn from Mendel and Weismann to the practical question--are acquired characters transmissible? On the one hand, are the virtues, which we laboriously assume, passed on into the natures of our children; on the other, are the sins of the fathers literally to be visited on the children unto the third and fourth generation?

This is the rock on which the old and the new schools of heredity are apt to split. From time immemorial, it has been thought that characters acquired during the life-time of the individual can be transmitted to the offspring; some have even gone so far as to believe that gross injury to the body may in some way be reproduced in the next generation. How far this is true is a question of supreme importance to the philanthropist, the statesman and the doctor.

It is necessary to define what we mean by an acquired character, and to do this we may use Weismann's own definition:

"Acquired characters are those which result from external influence upon the organism, in contrast to such as spring from the constitution of the germ"; and again, "modifications which are wrought upon the formed body in consequence of external influences."

To give a few illustrations of such acquired characters: The dwarfing of Japanese trees, changes induced in plants by transplantation into different climatic conditions, the enlargement of the lower lip or of the ear lappets, artificially produced among some savage tribes, the highly developed muscles of the blacksmith, the atrophy of certain muscles induced by constriction or other means in some Oriental beggars, or the contracted foot of the Chinese lady.

Is there any evidence, or any possibility of such acquired modifications being transmitted to the germ plasm from which the next generation springs?

We must eliminate from our minds all cases where the characteristic is germinal, not acquired, where it is due to a definite inborn variety, either a spontaneous germinal variety, whose transmissibility is unquestioned, or a germinal variety induced, as in animals and plants, by careful artificial selection. Some horses are bred for trotting, others for racing; some poultry for laying eggs, others for fattening. Individuals showing these desirable varieties are carefully separated and crossed, and their inherited tendency to these specialities is unquestionably heritable.

A good cricketer may be the father of a good cricketer, not because he has acquired the art of bowling and batting, but because he has the correct eye, the exact co-ordination between the eye, hand and brain, which was bred in him, and which he may transmit to his son; nor must we forget the force of tradition and of example, which may technically be called the liberating stimulus which influences the son to follow in the footsteps of his father.

The question is far more subtle than this. Suppose you took an average city clerk, with no inbred facility of eye or hand, and laboriously trained him to become a good cricketer, and he ultimately became the father of a son. Would that son have a better chance of having the special

inborn gifts which make a good cricketer, than if his father had stuck to his desk?

If the answer is in the affirmative, we must believe that the body cells of the parent can easily and intimately affect the germ cells which are the potential offspring, affecting them in such a manner that a result similar to that produced on the parent is produced in the germ cell.

As I said before, neither Weismann nor any other modern investigator affirms that prolonged bodily alteration in the parent cannot interfere with the well-being of the germ cell, even though we admit that the germ cell has a somewhat independent existence, and is not easily alterable; but this applies more to serious physical alterations, to chemical changes in the blood-stream which permeate the system and interfere with the vitality of the germ plasm, or, more satisfactorily, alterations which improve the bodily vigour of the parent and therefore improve the food supply on which the germ plasm depends; but that acquired character-bearing alterations, or physical alterations which do not affect the nutrition of the body of the parent, should influence the germ plasm is very hard to believe, nor is there any evidence to prove that such an effect is ever produced.

"The race has evolved protection for itself from individual excess," says Prof. Pearson, "by rendering the germ cells largely independent of the somatic (or body) cells and their changes."

Some experiments have been carried out to test the possibility of acquired bodily modifications being transmitted. Prof. Weismann cut off the tails of mice in twenty-two successive generations, but of the 1592 offspring from these curtailed parents not one showed any tendency to a diminished tail.

Dog breeders have had the custom for many years of biting off the tails of fox terrier puppies, and it might be supposed that Nature would have taken the hint and produced puppies ready born without tails; but nothing of the kind has happened; the unfortunate puppies have to submit to the unpleasant process just as their remote ancestors did.

Some experiments have been made with Alpine plants, which those who believed in the transmission of acquired characters at one time thought proved their case. These plants have been brought to southern gardens and have acquired characteristics new to their species, and their descendants grown in the same conditions have shown the same new variety. But, when these new forms were transplanted into poor soil, and the condition of their ancestors was thus restored, they reverted to the original type, showing that the modifications were not inherited, but were re-impressed by similar conditions.

I need not point out to you that the mutilations and other physical alterations which some races have performed on themselves for countless generations have never been inherited, but must be acquired *de novo* by each individual.

Other experiments, carried out on beetles, shrimps, and butterflies, etc., have gone to prove that marked change in climate or nutrition may produce germinal variations which are heritable, but such variations cannot be classified as acquired characteristics, as they originate in the germ plasm, and, like the Alpine plants, they tend to revert to the original form when the original conditions are re-imposed.

But, even if we must believe that acquired characters are not transmissible, there is still no ground to be despondent on the subject, nor to be unduly elated. It is no argument against the necessity of doing our very utmost in the training and education of the race, and of improving the conditions of life to counteract bad breeding. Without the opportunities which health and education give, even an inborn good quality may never have the chance of asserting itself. But we must learn to value good breeding more than we do.

There is a tendency in all democratic communities to consider one man as good as another, but putting aside all foolish shibboleths of wealth and rank, there remains the fact that sterling worth in the parents produçes a higher stamp of civic worth in the offspring. "Do men gather grapes of thorns, or figs of thistles? Even so every good tree bringeth forth good fruit; but a corrupt tree bringeth forth evil fruit." "We cannot get away from our inheritance."

At the same time, it must be borne in mind that good breeding includes the power to respond to good influences and environment; without good nurture the breeding may never find expression, whilst no such stimulus can produce as good results where the breeding has been inferior.

Again, as Prof. Thomson points out, "Although

what is acquired may not be inherited, what is not inherited may be acquired."

A better understanding of heredity leads to a greater tolerance and a wiser method of dealing with those whose inherited qualities are anti-social, whose tendencies are evil. We have relied too one-sidedly in the past upon the value of education in improving the stock; what we have come to see now is the necessity of awakening the national conscience to its responsibility regarding the inborn qualities of the future of our race.

Let those of us who have the goodly heritage of worthy ancestors say with Sir Thomas Browne:

"Bless not thyself that thou wert born in Athens, but, among thy multiplied acknowledgments, lift up one hand to heaven that thou wert born of honest parents, that modesty, humility, and veracity lay in the same egg, and came into the world with thee."

August 16th, 1915.

The Treatment of Sudden Obstruction of an Artery by Early and Prolonged Massage.—The treatment of sudden arterial obstruction by early and prolonged massage was first described by Merklen in 1903, when he recorded a successful case, and similar observations have followed.

Dufour and Mantoux describe the case of a healthy reservist, æt. 32 years, admitted to hospital on December 1st suffering from commencing typhoid fever. On January 5th the orderly observed purplish patches on the left lower limb, although the patient had felt nothing abnormal. Ten minutes later he complained of vague pain throughout the limb and some difficulty in moving his toes. The patient was seen by the writer exactly an hour after the onset. The entire lower limb was cyanosed and œdematous, especially in the toes. The limb was cold, especially in the foot. Pulsation was absent in the dorsal artery of the foot, the posterior tibial, and the popliteal, and even the femoral could be felt with difficulty at the groin. He complained of numbness and difficulty in moving the toes, but had no pain.

The limb was placed in the dependent position and energetically massaged along the course of the femoral and popliteal arteries and their branches. The massage was made from the centre to the periphery, and confined to the course of the arterial trunks, so as to force their contents to the periphery. In about 10 minutes the cyanosis disappeared, and the limb resumed its normal colour and became warmer. The numbness ceased and the movement of the toes became possible. The œdema persisted, and pulsation was still absent in the dorsalis pedis and posterior tibial. Massage was continued for 5 or 6 minutes at a time every half-hour throughout the day and the following night. The limb was maintained extended in a slightly dependent position, and enveloped in wool over warm fomentations. An injection of 0·2 grm. of sulphate of spartein was given, to increase the force of the blood-stream, and 3 grm. of citrate of soda to diminish the coagulability of the blood. On the following day the limb had regained its normal temperature, though the leg and foot were still slightly livid and œdematous. Pulsation in the femoral artery was now as strong as on the opposite side, but was still absent in the dorsalis pedis and posterior tibial. The patient had slight hæmoptysis, probably from small emboli in the lung. The massage was continued as before. Next day the œdema had almost disappeared, and pulsation was detected in the dorsalis pedis. Three days later pulsation in the foot was normal. There had been recurrence of the hæmoptysis and a congested patch was found at the base of the left lung. The patient's local and general condition remained satisfactory. The typhoid fever ran its usual course, and his temperature, which was unaffected by the local condition, became normal in the end of January. Two months after the onset he could walk a kilometre without fatigue, but showed slight œdema of the ankle after exertion. The posterior tibial artery remained permanently obstructed.

The nature of the arterial obstruction was doubtful, but whether thrombosis from arteritis or embolism, the massage probably dispersed the obstacle and secondary clot. The circulation of the whole limb was seriously impaired, and, but for the treatment, extensive gangrene would probably have followed. The value of this method was well demonstrated. Only on failure of massage should arterotomy and removal of the clot be attempted. This operation has been successfully performed, but, in this case, it would have been difficult to determine the best site for the incision. —*Bulletins de la Soc. Méd. des Hopitaux*, p. 143.

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We regret that owing to the war the number of pages must be diminished for a time.

FOUR GRESHAM LECTURES ON HEREDITY.

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

LECTURE II.

THE SOCIAL ASPECT OF HEREDITY.

MENDEL'S law, formulated from experiments in the crossing of plants, is widely applicable to animals and plants; it appears to hold good in certain inherited physical qualities in man, and the more accurate study of human pedigrees is likely to prove that the inheritance of mental and perhaps artistic faculties harmonises with it.

Weismann's microscopical investigations, which have brought to light the now generally accepted belief in germinal continuity and the comparatively independent life of the germ plasm, go far to prove that acquired characteristics, valuable as they may be for the welfare of the race, cannot be directly transmitted to the offspring; that we cannot get away from our inherited moral and physical character; that nature is far stronger than nurture. The breeders of horses and dogs do not question which of these forces is the stronger, in the production of the type they desire. Good training will develop a character; but only if the pedigree has first implanted it.

Let us follow the teaching of Sir Francis Galton, who attacked the subject by the statistical method, and who is the founder of the new science, which he named "Eugenics." This is the science which deals with the influences which improve and develop the inborn qualities of a race; it is the science which serves to disseminate accurate knowledge regarding them, and encourages action in the hopes of creating a higher standard of race efficiency.

Francis Galton was born in 1822. On his father's side he came of a Quaker family of bankers

in Birmingham; but both his father and his grandfather were men of science by inclination, if not by profession. His mother's father was the well-known Dr. Erasmus Darwin, physician, philosopher and naturalist. Erasmus Darwin was twice married, and was grandfather to two illustrious descendants, the immortal Charles Darwin, grandson by his first wife, and Francis Galton, grandson by his second wife. Galton's education was conducted on strictly classical lines at King Edward's School in Birmingham, till he left at the age of sixteen to begin his studies in medicine. Unlike the medical student of the present day, who has to assimilate a vast store of theoretical knowledge before he gains much practical experience, Galton entered the Birmingham General Hospital at once as an indoor pupil. He found the work congenial, and after a short time he passed on to King's College in London, where he attended scientific lectures, and studied medicine at King's College Hospital.

At the age of eighteen he went to Giessen in Germany, to listen to the lectures of Liebig, the chemist; but finding that he was unable to benefit by his teaching, owing to his ignorance of chemistry and German, he started off bravely to travel alone, all over Europe, as long as his money lasted. After some months he returned to England and entered Trinity College, Cambridge, where he studied mathematics and medicine, and made many life-long friends. From there he went to St. George's Hospital in London, but finding on his father's death, in 1844, that it would be unnecessary for him to take up any profession, he abandoned his medical studies and began further travels to Egypt, Syria and elsewhere; and he penetrated to the interior of South-West Africa, where he successfully carried out some exploration. On his return to London, he interested himself in meteorology (he is the author of the word we hear so much quoted now, "anticyclone"), in geography, in anthropometric work and other studies relating to heredity. He expressed his views regarding race improvement in two articles contributed in 1865 to 'Macmillan's Magazine'; and in his book 'Inquiries into Human Faculty,' published in 1884, he first introduced the word "eugenics." In describing one of the objects of the work, he said: "Its intention is to touch on various topics more or less connected with that of the cultivation of race, or, as we might call it, with eugenic questions." In the introduction he says: "My general object has been to take note of the varied hereditary faculties of different men, and of the great differences in different families and races; to learn how far history may have shown the practicability of supplanting inefficient human stock by better strains; and to consider whether it might not be our duty to do so by such efforts as may be reasonable, thus exerting ourselves to further the ends of evolution more rapidly and with less distress than if events were left to their own course."

From this time onward he devoted his energies to furthering the knowledge of all subjects connected with race improvement, the far-reaching importance of which he realised before the rest of the world had woke up to it, and he succeeded in arousing considerable interest in the subject among cultivated people at home and abroad. "I kindled the feeble flame," he wrote much later, "that struggled doubtfully for a time until it caught hold of adjacent stores of suitable material and became a brisk fire, burning freely by itself."

In 1904 he founded a Research Fellowship at the London University, to which post Mr. Edgar Schuster was appointed, and a research scholarship was added, which was filled by Miss Elderton. Prof. Karl Pearson is now Superintendent of the Eugenics Laboratory, Dr. David Heron is the Research Fellow and other posts have been added.

On Sir Francis Galton's death in 1911, he bequeathed his residuary estate to form a permanent endowment of the laboratory, which exists entirely for research work; whilst the Eugenics Education Society, founded in 1908 with Galton as first Hon. President, concerns itself with the dissemination of facts and ideals bearing on eugenics. Major Leonard Darwin, Galton's cousin, is the acting President, and his inspiring lectures should be heard and read by anyone fortunate enough to get the chance.

To Galton's careful statistical work, which has been mostly carried on by Prof. Karl Pearson, we are indebted for much additional and more exact knowledge of heredity; and I may mention two laws only which he has formulated. The first has been named the Law of Ancestral Inheritance; that is, the share which the various ancestors have, on an average, in the inheritance of any individual organism. "The two parents," he says, "contribute between them on the average one half of the total heritage of the offspring, the four grandparents one

quarter, the eight great-grandparents one eighth, and so on." Thus the mean character of the individual can be calculated with the greater exactness the more we know of the characters of his ancestry. Galton worked out his conclusions on data as to stature and other qualities in man and as to the colouring of basset hounds. "Looked at from the social standpoint," says Prof. Pearson, "we see how exceptional families by careful marriages can within even a few generations obtain an exceptional stock, and how directly this suggests assortative mating as a moral duty for the highly endowed. On the other hand, the exceptionally degenerate, isolated in the slums of our modern cities, can easily produce permanent stock also; a stock which no change of environment will permanently elevate, and which nothing but mixture with better blood will improve. But this is an improvement of the bad by a social waste of the better. We do not want to eliminate bad stock by watering it with good, but by placing it under conditions where it is relatively or absolutely infertile."

The second generalisation contributed by Galton, which is a valuable contribution towards the study of heredity, is the Law of Filial Regression, which he based on the records he made of family faculties and his anthropometrical measurements. The records dealt chiefly with stature, eye colour, temper, artistic faculty and some forms of disease. The law formulates what had previously been a mere impression, *i. e.* that the children of exceptional parents (for good or ill) tend to be more commonplace than those parents, that is, they revert to the average. "The law of regression," to quote Galton's own words, "tells heavily against the full hereditary transmission of any gift. Only a few out of many children would be likely to differ from the mediocrity so widely as their mid-parent, and still fewer would differ as widely as the more exceptional of the two parents. The more bountifully a parent is gifted by nature, the more rare will be his good fortune if he begets a son who is as richly endowed as himself, and still more so if he has a son who is endowed yet more largely. But the law is even-handed; it levies an equal succession tax on the transmission of badness as of goodness. If it discourages the hopes of a gifted parent that his children will inherit all his powers, it no less discountenances extravagant fears that they will inherit all his weakness and disease." Filial regression is explained by the fact that the individual is the product, not only of his parents, but of a vast ancestry; and the mean of that ancestry is probably not far from that of the general population. "In the tenth generation a man has 1024 tenth-great-grandparents. He is eventually the product of a population of this size, and their mean can hardly differ from that of the general population."

Before applying the laws of heredity to individual characteristics, we will, to-night, examine their effect on certain social problems with which we are faced. It should be realised that these problems are comparatively new, because we are only now beginning to feel the consequences of the fact that man, in his incorrigible habit of interfering with the laws of Nature, has once more, and quite irretrievably upset the methods which she has pursued since time immemorial to effect race improvement; methods both drastic and cruel, but immensely effective.

Her scheme, in fact, was that of natural selection, and in primitive man, just as in animal life, she carried it out, and still carries it out, without let or hindrance, not sparing the individual, nor hesitating on sentimental grounds, but ruthlessly weeding out the inefficient by early death, whilst giving long life and a large issue to the strong and the capable. Through this means she has slowly and surely worked for a steady racial improvement.

It is sometimes still asserted that natural selection cannot be seen at work; and that, even if that power existed, it no longer persists. But how otherwise can we account for the extinction of primitive races, a process which is going on all the time, and which is deplored by sentimentalists as if it were the work of brutal man, purposely elbowing out the native of a country from his birthright? It is nothing of the kind, at least when a civilised people colonises a country inhabited by a primitive race; for the advent of new races, such as the British in New Zealand and South Africa, the Boers in South Africa, and the Europeans of different nations in the United States, is not carried out by wholesale massacre or the deportation of the existing population; but the work of extinction goes on quietly, slowly and quite naturally, through the impossibility of the lower race competing in the conditions imposed upon it by the dominant people. The world is permanently enriched by such supplant-

ing; whereas when the inferior stock (as in hyper-civilised and democratic communities) is bolstered up and assisted in elbowing out the superior by the sheer weight of its numbers, a condition of things is established which threatens disaster.

It must be kept in mind that the immigration and colonising of an alien race only take place on a permanent basis where the native population is inferior; and successful immigrants are always people of some strength of character, which is a fact constantly brought before social workers. The gentle, submissive man endures and succumbs; it is the man of spirit and enterprise who, feeling that his faculties are greater than his opportunities in his native country, has the courage to transplant himself and seek for a wider scope for his energies. It is from this strain that a new race and a dominating people spring.

The beneficent work which Nature is for ever striving to carry out, with a ruthless disregard for the individual, and to the great benefit of the race, can no longer be openly permitted among civilised communities. The whole strength of popular opinion, showing itself in unbounded charity, in legislation, and in science, is pitted against Nature's efforts to weed out the weak and incapable. The strong have shouldered the burden of their weaker brethren with a splendid disregard for their own security, but alas! without realising that they are not permanently benefiting even those they wish to help; for each generation sees the growth of the weeds of incapacity.

I hope, later on, to speak of such powers as still lie in our hands to check the evil; but lest the cause of eugenics should, if only for a moment, be misundertood, let me hasten to say that eugenists only plead this: though all men, the most worthless, the diseased, and the deficient included, have the right to live, it is not so obvious that they have the right to reproduce their kind and perpetuate their affliction.

"If," as Prof. Karl Pearson writes, "we check Nature's effective, but roughshod methods of race betterment, we must take her task into our own hands, and see to it that the mentally and physically inferior have not a dominant fertility." Here we come back to the question of heredity.

It is admitted by all that like produces like, that the mental and physical character of the parents is inborn in the children; but legislation has built all its plans for race improvement on the belief that improved surroundings will produce a better breed of man; it has absolutely disregarded the fact that Nature, now, as ever, is far stronger than nurture. Who, admitting this, can view the enormous birth-rate in the very lowest social classes, and the falling birth-rate among the educated, without a sense of disquietude? Yet the whole trend of legislation is to encourage the fertility of the improvident, the shiftless and the lazy, by relieving them of the burden of their parentage, and casting this burden, with a reckless disregard of consequences, upon the shoulders of the prudent and hard-working. An answer was given lately, in reply to the question, What is a statesman? "A statesman thinks of the next generation, while a politician thinks only of the next election."

Free meals, free education, cheap trams, the limitation of the hours of labour, providing leisure to watch two football matches a-week, with a minimum wage and free medical advice will bring votes to the demagogue; but will they solve the problem of the ever-increasing burden of taxation, compelling the prudent to limit their families? The voluble platform speaker has no time to study the root causes of our social burdens.

For three generations or more, social legislation has worked for the betterment of conditions under which man lives, and has worked nobly, cleaning out the foul places of great cities, bringing improved sanitation into the meanest streets, seeking out disease, bringing education within the reach of every child, protecting the mother and the infant. Work of this kind must continue, as also the great part played by organised charity, which tends to the betterment of human life by giving the worthy a chance of showing his capacity; but the most sanguine social worker cannot persuade himself that these mighty efforts at human betterment are, even in the faintest degree, checking the number of unemployables and inefficients in our midst, nor is there the slightest prospect, if we go on as we are doing, that we shall reach a time when all England will contain only healthy and self-supporting men and women.

To divide the community into worthy and unworthy according to wealth or poverty is unsatisfactory; but it must be admitted that the greatest number of unemployables, feeble-minded, paupers, alcoholics, criminals and all such as constitute the

dead weight of the population, which has to be supported by the rates, springs from the very poorest and lowest people, and it is this class which constitutes the super-fertile element of the population.

Very careful statistics bearing on this question have been drawn up by the Eugenics Laboratory. We have become accustomed to reading the fact, recorded with unfailing regularity by the Registrar-General in the public press, that the birth-rate of each week is so much below the average number in the corresponding weeks of the previous five years; and even that it is the lowest rate since registration began. This decline has been persistent for thirty-seven years in France, which has a lower birth-rate than any other nation. A situation was reached in 1911 which might be called disastrous; the excess of deaths over births in that country having reached the appalling figure of 34,869, instead of an excess of 70,000 births over deaths in 1910. This excess of deaths over births was still present in 38 out of the 87 departments in 1913, but the birth-rate over France generally was slightly better. It is a lamentable fact that, in less than forty years, the birth-rate of France has diminished by 200,000 a year.

If she cannot find means to stop this terrible condition of things, this splendid nation, with all her wealth of genius, must look forward to being overwhelmed by more fertile races seeking for elbow room and to ultimate extinction.

Now, how does England stand in comparison?

Our birth-rate is now down to the level of France thirty years ago, and there are many signs that there will be a further and continuous fall. The causes are said to be numerous, but the State should make an effort, by encouraging larger families among the well-to-do and able section of the population, to check this danger. The better the position a man has gained for himself, the more he shrinks from seeing his children compelled to take up a lower standard of living, forced upon them by poverty. Increased taxation, increased cost of living, and a government never as anxious to help the prudent as to support the improvident, compels the conscientious man to marry late and have few children, determined as he is to sacrifice his own inclination for the sake of improving their chances. But the improvident, the selfish and the man who has learnt to look to State support as his birthright, are not influenced by any such deterrents.

Thus, the people with the lowest intelligence in the population have the highest birth-rate, and those with the highest intelligence have the lowest birth-rate.

"Approach the problem as we will," says Prof. Pearson, "the conclusion forced upon us is ever the same; the physically inferior, the mentally slow, are not naturally more fertile than the stronger in body and mind, but they are in our community to-day the more fertile. The moment we suspend the full vigour of natural selection, the moment we artificially correlate fertility with any defect of physique or intelligence, we start a downward movement."

These people of the class under consideration marry early, boys and girls of eighteen or twenty, they have large families, and though the death-rate among their children is higher than among the better classes, yet their relative fertility is so much greater, and the generations succeed each other so rapidly, that in the end they are responsible for the largest share of the next generation.

The professional classes marry late, and in England the marriage rate of this class is probably not more than half that of the industrial class, with the result that "society recruits itself from below, the rate of the industrial classes being 30 per cent. greater than that of the professional classes."

In the study of the inheritance of human characters, it was reckoned that 52 marriages in 10,000 are of an exceptional man and woman; the remainder are not. From these 52 exceptional people about half the children are exceptional. Of commonplace parents rather less than one twentieth of the children will be exceptional.

Sir Francis Galton tried to make an estimate of the worth of children, and reduced it almost to a mathematical formula.

"The brains of the nation lie in the higher of our classes." Man has evolved from a lower type to his present dominating position in the world through the superiority of his brains over the lower animals. The struggle for existence in him has not been a struggle of physical force, but in cunning and skill, in which he outwitted his competitors. The struggle continues, and the man with superior brains can rise in spite of adverse circumstances, in spite of nurture, if Nature has implanted the right stuff in him. The clever man rises to the top and begets children who inherit some of his

qualities, and we must therefore look for the brains among the upper classes.

The least valuable human beings are criminals, loafers, casual labourers, drunkards, a class described by Mr. Charles Booth thus: "Their life is the life of savages, with vicissitudes of extreme hardship and occasional success. From them come the battered figures who slouch through the streets and play the beggar or the bully. They render no useful service, they create no wealth, more often they destroy it. They degrade whatever they touch and as individuals are perhaps incapable of improvement. They are barbarians, but they are a handful." Galton points out that many who know these people say that it would be an economy and a great benefit to the country if all habitual criminals were rigorously segregated and denied the right of producing offspring, for there is no doubt that the criminal character is hereditary to a very considerable extent.

The lessened fertility of the provident classes is artificial and is greatly forced upon them by circumstances. The lowest people increase at the expense of the more worthy.

But in order to furnish more exact data concerning the reasons of the declining birth-rate, a Commission has been formed consisting of distinguished members of various churches, doctors, and other representative men and women. A form of questions has been evolved and is being privately circulated among certain educated classes. The Commission will inquire into the extent and character of the declining birth-rate, its causes, its effects and its economical and national aspect.

To quote again from Mr. Patterson's account of the riverside population of South London: "The dull-witted or imbecile, the epileptic and alcoholic marry and are given in marriage, while the nation stands by, building institutions and ruling out columns of figures for the sad result. A man, æt. 30 years, had drunk heavily for years and was at last struck down by alcoholic paralysis of the brain and compelled to give up his work. He married a young girl, and became the father of thirteen children. Neither Church nor State had the power to forbid this unholy union, and now half a dozen different authorities, penal, medical and philanthropic, bear the burden of the seven children who survive. It is no interference with human liberty to stay the hand of the man who strikes a living woman or a child, yet as a nation we hesitate to thwart the man or woman who, by their wantonness, are killing or maiming the children yet unborn."

Such an extreme case points the moral once more that, for the betterment of the race, not improved environmental conditions, nor well-run asylums or hospitals, nor the ever-ready help of charity, will touch the root of the evil. Inquiries into the pedigrees of alcoholics, criminals, prostitutes and unemployables, reveal the fact that in an enormous majority of the cases these individuals are themselves moral or mental defectives and are the descendants of mentally deficient parents. How this defect originally arises it is hard to say, but it appears to be a distinct germinal variation, and as such is heritable.

Sir Clifford Allbutt writes: "I regard feeble-mindedness (if not accidental) as always hereditary. I have never met with a case of manufactured feeble-mindedness apart from some accident either at birth or soon afterwards. All arrested development is apt to run in families."

Under the heading of feeble-minded or defective persons in England must be classed, not only the lunatics in asylums, but about 200,000 persons who, "though not certified as insane, are yet not in such sufficient possession of their faculties as to be capable of properly looking after themselves." From almost every civilised country statistics show that this huge host of degenerates is increasing from year to year. "In the ruder state of society which has passed away," writes Sir Edward Fry, "little heed was taken of these unfortunate children, and many of them, no doubt, died comparatively early in the struggle for existence. But we have learnt to think more tenderly of the inferior members of our race; and we seek to protect them from the calamities and sufferings to which they are naturally exposed, and to preserve their lives to the utmost. But in so doing, and so doing rightly, we incur, it appears to me, another responsibility, namely, that of preventing, so far as we reasonably can, the perpetuation of a low type of humanity; for otherwise the munificence of one generation becomes the burthen and the injury of all succeeding ones. . . . The ranks of the insane as well as the imbecile are recruited from the children of the feeble-minded."

Defective persons tend to have much larger

families than normal people, for their utter lack of forethought and control puts no limit to the number of children born, and it has been found that their families average 7·6 children, whereas the normal families average 5. In other words, 20 defective parents are likely to produce 76 children, while 20 normal parents will only produce 50 children.

It is found that 82 per cent. of criminals and defectives come of defective stock; for though the children may not show the same form of mental defect, yet among them are found the majority of incorrigible rogues, alcoholics and many epileptics. Like more normal people, they appear to be attracted to their own type; and no normal child, it is said, has ever sprung from two defective parents.

Under the system which has prevailed till now these unhappy people have drifted irresponsibly through their wretched lives, and we can trace their career, from uncared-for childhood into the ranks of casual labour, from which they drift down and ever downwards into petty crime, immorality and so into prison and penal servitude. "One investigator only found from 4 to 5 per cent. of criminals whose parents were really sound." No reformatory methods in prisons or institutions have any permanent effect on them. They are easily influenced, but as easily corrupted.

A Royal Commission was called in 1908 to consider the existing methods of dealing with defectives of all grades, "in view of the hardship or danger resulting to such persons, and to the community, from insufficient provision for their care, training and control; to report as to the amendment in the law and other methods which should be adopted in the matter." Their report, drawn up after elaborate investigations, shows how little benefit these unfortunates have derived from the present system which has so far dealt with them. "On general grounds," it says, "many competent observers are of opinion that, if the constantly recurring, fatuous, and irresponsible crimes and offences of mentally defective persons are to be prevented, long and continuous detention is necessary. . . . From the earliest age, when they appear before the magistrates as children on remand, or as juvenile offenders, until and throughout the adult period of their lives, the mentally defectives, at first reprimanded and returned to their parents, then convicted and subjected to a short sentence and returned to their parents, and then continually sentenced and re-sentenced and returned to their parents or friends, till, for crimes of greater gravity, they pass to the convict prison, are treated, as this reiterated evidence shows, without hope and without purpose, and in such a way as to allow them to become habitual delinquents of the worst type, and to propagate a feeble-minded progeny, which may become criminal like themselves. This is an evil of the very greatest magnitude."

The workhouse authorities know the type well—the unemployable who drifts in and out, a perpetual charge to the parish, the vagrant, the tramp, and the unhappy girl who comes in to give birth to her illegitimate child, which probably inherits her degeneracy and returns year after year for the same purpose. Havelock Ellis tells us that: "It has been found that of nearly 15,000 women who passed through the Magdalen Homes in England, over 2,500, or more than 16 per cent.—and this is probably an under-estimate—were definitely feeble-minded."

The general effect of the evidence before the Commission on the causation of mental defect is summed up thus: "(1) That, both on the grounds of fact and of theory, there is the highest degree of probability that 'feeble-mindedness' is usually spontaneous in origin, that is, not due to influences acting on the parents; and tends strongly to be inherited. (2) That, especially in view of the evidence concerning fertility, the prevention of mentally defective persons from becoming parents would tend largely to diminish the number of such persons in the population. (3) That the evidence for these conclusions strongly supports measures, which on other grounds are of pressing importance, for placing mentally defective persons, men and women who are living at large and uncontrolled, in institutions where they will be employed and detained, and in this and in other ways kept under effectual supervision so long as may be necessary."

It cannot be too persistently reiterated that the study of social evils convinces us of the fact that, in the past, philanthropy and legislation have depended too much upon the improvement of the conditions of life, and have not given sufficient attention to the origin of life itself. A better knowledge of heredity is absolutely necessary to tackle the evils of to-day, and there are signs that this necessity is forcing itself upon intelligent men and women; that the social worker, and those

statesmen who desire more to benefit mankind than to increase their own popularity, are trying to base their efforts on the conviction, which cannot be too often repeated, that Nature is, and always will be, a stronger force than nurture.

August 23rd, 1915.

A System of Clinical Medicine; Dealing with the Diagnosis, Prognosis, and Treatment of Disease; for Students and Practitioners. By Thomas Dixon Savill, M.D.Lond. Fourth edition. London: Edward Arnold, 1914. 25*s*. net.

The third edition of this book appeared in 1912, and the fact that two years later another is called for indicates that it supplies a large and growing demand. Its peculiar features are well known. It approaches the subject from the standpoint of symptomatology, so that the principle throughout is to trace from symptoms the underlying morbid cause. The habit is valuable in itself, as it develops and perfects the faculty of acute and accurate observation. The book, while a lineal descendant of Fenwick's 'Medical Diagnosis' or Finlayson's 'Clinical Manual,' expands and applies liberally all the best features of these early and successful works. While supplying the busy practitioner with the materials for ready and differential diagnosis, it also caters for the doctor who is leisured, and who can apply to his cases the many aids furnished by recent laboratory methods, and research in clinical bacteriology. No clinical features are too minute to be overlooked, and possible errors and pit-falls in diagnosis are wisely and at some length plainly pointed out. The chapter on "Diseases of the Heart" has been recast, while revision of certain subjects has been undertaken by experts in the different subjects. The book contains many excellent and artistic illustrations specially designed and taken off actual cases. This edition should have the same success which has attended the former ones.

A Text-book of General Embryology. 10*s*. 6*d*. net. Outlines of Chordate Development. 10*s*. 6*d*. net. By William E. Kellicott, Professor of Biology in Soucher College. London: Constable & Co. 1914.

The two books might in a sense be regarded as complementary. In the first—the result of the direct outgrowth of experience as a collegiate study—the general principles of the science of life are approached from the standpoint of their general biological relations. The selection of the facts, the method in which the subjects are discussed, and their presentation, indicate that the student, as such, has been first in mind. The book is a text-book rather than a hand book, and the arrangement of the subject matter is such that, by a difference in type, he may take a longer or shorter course of study.

In the second book the student is introduced to the study of chordate development through the embryological medium of the "amphioxus." Then follows a full account of the development of the frog, and subsequently of the chick. In the final chapter on the "Mammal" are included the earlier stages of embryo formation, the establishment of its relation with the maternal organism and the development of the external form of the human embryo. Both books are profusely illustrated, so that the building-up process can be traced step by step. Some of the illustrations might profitably have been coloured.

Medical Nursing. By A. S. Woodward, M.D. London: Edward Arnold. 1914. 4*s*. 6*d*. net.

This manual might be claimed jointly by the nurse and the medical practitioner. As Dr. Woodward points out, the nurse has evolved and "become an educated co-operator," with corresponding aspirations which must be satisfied. Hence the necessity for such a book. The chapter on the "Administration and Action of Medicines" is well written—clear, definite, and terse. There are excellent chapters on the "Nursing of the Insane" and on the "Nursing of Sick Children." The "Care of the Dead," "Dying Declarations and Will-making," contain instructions all nurses should know. There is an appendix of "Emergencies," with many useful points, both on what to do and what not to do. The book contains about seventy illustrations. We commend it to the trained nurse and the young practitioner.

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We regret that owing to the war the number of pages must be diminished for a time.

FOUR GRESHAM LECTURES ON HEREDITY.

By F. M. SANDWITH, M.D., F.R.C.P.
Gresham Professor of Physic.

LECTURE III.

THE INFLUENCE OF HEREDITY IN DISEASE.

THE study of the influence of heredity in the production and persistence of definite characteristics is one of very special importance to the practising physician, who has to take heredity into almost daily account in his efforts at accurate diagnosis and prognosis.

It is common knowledge to anyone who has applied for a policy of life insurance that a good or bad family history modifies the opinion of the examining doctor as to the value of the candidate's life.

Until the discoveries of Weismann and others had established the fact that what is inherited is only what is contained and carried by the nuclear chromatin of the reproductive germ cells, there was a good deal of loose talk about inherited disease, which became a veritable nightmare to those who had relatives who had died of diseases supposed to be heritable. But now that we have come to understand not only the principle of germinal origin, but also that most diseases are carried by the presence of definite micro-organisms in the patient, it becomes obvious that it is extremely improbable that such micro-organisms (such as the leprosy bacillus or the tubercle bacillus) could invade the germinal matter. Even if they could do so they would not, strictly speaking, be a part of the heritable qualities of which the germ-plasm consists; though for all practical purposes this fine distinction between what is inherited and what is transmitted by poison in the germ plasm is of comparatively little interest. It is certain that diseased conditions,

which appear mostly in the offspring at birth or soon after, may arise from germinal variations induced by a diseased condition of one or both parents; but these, and other diseases produced by infection before birth through the blood-stream of the mother, cannot be classified under inherited diseases, for they are not transmissible to future generations; they are the product, in the individual affected, of germinal infection only. True inheritance is only what is inherent in the germ cell before fertilisation.

Inherited morbid phenomena, which are transmissible, being often very persistent through many generations, are distinct from these, and include (1) gross deformities; (2) anomalies of defect; (3) susceptibility to specific infection; and (4) lack of nervous stability.

Gross deformities arise in animals and plants as sports or variations; and in many cases this is likewise the case in man, though in his case, owing to the slow rate at which each generation is produced, accurate knowledge is often difficult to obtain, and what appears to be a sudden new variety may be an inheritance from a distant ancestor, an inheritance which has remained latent for some generations.

Sports occurring in animals have sometimes been turned to advantage by man, who has, by careful breeding, produced new permanent varieties by so doing. Hornless cattle have been so bred, tailless cats and short-tailed dogs and pigs, and the famous royal cream white horses of Hanover have been produced by very careful inbreeding from a single "sport," a white horse which appeared in a strain which had previously produced no white horses. Here, in the words of Darwin, we see "the will of man" acting in the place of natural selection, as he "accumulates the variations in certain directions."

In Nature the causes of variation are still obscure, but the fact remains that there is a constant tendency to slight variation in all living things, which may in part be due to the influences of the environing body on the germ cells, to the mingling of ancestral inheritance, or to some arrested development; the latter being clearly the case in certain malformations, such as hare-lip.

But, besides slight variations, sudden varieties or sports occasionally arise; and when they do they are very persistent, those possessing the abnormality appearing to be the dominant type, the majority of their offspring sometimes exhibiting the peculiarity.

Achondroplasia is a variation which appears to be very hereditary. In former days these dwarfs, handicapped from birth, rarely grew to maturity and the females seldom survived the birth of a child. Our humanity has, very rightly, caused the survival of these poor, deformed creatures and surgical skill even enables them to become the mothers of several offspring.

Another gross malformation, popularly called lobster claw, where the individual has one finger only on each hand, has, from a pedigree put together by Professor Karl Pearson, been inherited by 25 descendants out of 75 from one deformed woman.

It is very generally believed that outward malformation often coincides with mental abnormality; a healthy mind rarely inhabits a body grossly deformed from birth. It is no use relying upon legislative interference to stop the production of monstrosities; there are limits to official powers. Unfortunately it is equally difficult to reach by popular opinion the human strata from which most of these cases spring.

Polydactylism, or the presence of additional fingers or toes, is more common, and has been noted in six generations of a human family; even the slight check to reproduction which may arise through a physical repulsion from people so affected is now rendered nugatory by the successful amputation of such parts, which enables the affected individual to resemble the normal.

Minor malformations, such as peculiarities in dentition, or the shape of ears, are common and hereditary.

Among the anomalies of defect in which heredity plays an important part may be mentioned the interesting disease, hæmophilia. There is a popular belief that it is caused by the individual affected having one skin less than most people; in fact, I have heard it said that most of us have seven skins, and these unfortunates have only six!

The cause of hæmophilia is still a little obscure, but appears to be due to a deficiency in the blood. When bleeding takes place from a small wound the blood tends to coagulate, forming a natural plug which stops up the wound. But in hæmophilic people it is now generally believed that there is a defect in the coagulating power of the blood,

which is the reason for the immoderate hæmorrhages which are such a constant danger to them.

Serous effusions also invade their subcutaneous tissues and joints, causing huge bruises and swellings, and these occur either as the result of quite trifling injuries or spontaneously, and great pain may follow. The disease is worst in childhood and is less marked as the boy grows older; it is nearly always of hereditary origin, and it belongs to a peculiar group called sex-limited diseases, appearing only in the male sex. Boys with hæmophilia are very difficult to rear. The slightest blow or fall, or even a strain, will cause a joint to fill with serous blood which does not become absorbed as in normal people, but the joint remains swollen and water-logged, crippling the patient. This result is most often seen in the knees, ankles, or elbows. Nose bleeding of an alarming character, bleeding from the gums or internal organs, may continue till the patient dies from exhaustion.

The extraction of a tooth, and even a very slight surgical operation, are therefore attended by the gravest danger to life.

Although the bleeding may be checked after some hours, it is liable to begin again when the patient falls asleep; and Sir Almroth Wright, in describing the disease, says:

"In such cases, if the boy sleeps on uninterrupted and no one watches over him, the bed may be soaked with blood until it drips upon the floor. The perfectly desperate condition that things get into in the houses of the poor when such bleeding as this occurs is more easily imagined than described. I have seen a room look like a shambles, and I know a case where the mother, finding her boy bleeding in his sleep, had to wrap him in the blood-soaked blanket and to wheel him on a coster's hand-cart in the middle of the night, bleeding all the way, from a suburb to one of the London hospitals."

The outlook in such patients used to be practically hopeless, but modern therapeutics have greatly lessened the danger to life, though the crippled joints and the constant fear of hæmorrhage must make the life of the unfortunate victim of little joy to him.

The hereditary tendency of this disease is what chiefly interests us now; and the gravity of this question is increased when it is realised that the women in families so afflicted are peculiarly fertile, and it is through the women that the disease is conveyed. Women who are the daughters of bleeder-fathers do not show the inheritance, at least in the acute manner that is apt to occur again in their sons, though many peculiarities may be detected in their blood. But the disease appears to be latent in them, to find expression in their male offspring.

Another instance of a defect seen almost exclusively in males, but transmitted through the mother, is colour-blindness. The colour-sense is the power the eye has of distinguishing light of different wave lengths, which cause us to see different colours. The defect, when present, varies in degree from a total inability to distinguish between any colours to a condition in which there is only confusion between one or two colours.

Normal sighted persons can see six definite colours in the spectrum; the colour-blind see five, four, three, two or one colour, according to the intensity of their defect. This peculiarity is present in the population in the proportion of 3·5 per cent. in men, but only 1 per cent. in women. It may not be of great importance to most people whether their colour vision is abnormal or not, and if the defect is not pronounced they may remain unconscious of it all their lives. A distinguished engineer, a friend of mine, cannot distinguish red and green. He has travelled in many parts of the world, and has now decorated his walls with sketches he has made of the world as he has seen it, in which the colouring is all blue and brown. He has faithfully produced what he saw, and it gives him, but not his friends, great pleasure to look upon this curiously monotonous colouring.

No candidate qualifying for the navy, the army, some of the civil government services, or the mercantile marine, will be admitted if he is colour-blind. Women, as I have said, rarely show this defect, but, if a woman is the daughter of a colour-blind man, she may carry the defect and transmit it to her sons. If a woman is colour-blind, she is the daughter of a colour-blind father and of a mother who carries the defect from her father.

The accuracy of a pedigree is often difficult to arrive at because of the fact that, unless the colour vision is markedly wrong, or it happens to hinder the individual in the business of life, he may remain unconscious of it. When it occurs in a marked degree it appears, apparently, as a sport, but the

probability is that the parent or grandparent is really the culprit, having suffered from the defect to a limited extent.

When we come to inherited susceptibility to disease, we can all recall certain families among our acquaintance where there is either a tendency to "take" every infectious illness of childhood, and to take them in a severe form, or a tendency to one special disease, generally one of the forms of tuberculosis. In this latter case, the belief prevailed for a very long time that tuberculosis was directly inherited from parent to child, and the mistake arose owing to a want of knowledge regarding the causation of the disease and ignorance of the physical carriers of heredity.

Sir Thomas Watson, writing in 1871, asks: "Is phthisis contagious? No, I verily believe it is not." He believed that it was never imparted from one individual to another, but that to prevent the disease there should be "prudent avoidance of ill-assorted marriages" between people with the so-called "strumous diathesis." And he held firmly to this belief in spite of cases which he quoted where one sister nursed another dying of phthisis and presently "droops and dies of the same complaint," and again where a wife, with "no traceable or acknowledged example of scrofula in her pedigree," watching by the bedside of her husband dying of consumption, develops the dread disease and dies. "No," he says, "I am satisfied that the disorder does not spread by contagion." He considered that the mental anguish, fatigue, and the unwholesome sick chamber, "call forth the fatal malady." It never occurred to him, as it does to us now with our wider knowledge, that the ill-ventilated sick rooms of his days were veritable hot-beds of infection. Hirsch, in 1884, stated: "Of real importance for the frequent occurrence of phthisis is the transmission of the disease by way of heredity. That phthisis propagates itself in many families from generation to generation is so much a matter of daily experience that the severest sceptic can hardly venture to deny a hereditary element in the case."

Yet now, thirty years later, we do deny that phthisis is hereditary. Koch and others succeeded in demonstrating that tuberculosis, of which phthisis is the best-known variety, is due to the tubercle bacilli which are present in enormous numbers in the part of the body affected. It does not appear possible that the bacillus can invade the germ plasm which is the potential offspring. What may occur is that the bacilli are transmitted to the unborn child through the blood-stream of the mother. This is infection before birth, not heredity, and even this is very rare. The children of parents even actively infected by tuberculosis are generally born free from infection, and are often surprisingly vigorous, as if to prove the independent existence of the germ cell from its surroundings. But undoubtedly the majority of children born of diseased parents come into the world ill-nourished and weakly, because the toxins in the body of the parent must prejudicially affect the blood-stream, on which the nourishment of the germ plasm depends. To this extent, therefore, heredity plays a part, and a very important part, in the transmission of disease; though the actual disease germs cannot, it seems, in the case of infectious illnesses, be inherited. What is inherited is a weakened constitution which is unable to resist the attack of its microbial enemies.

To this must be added the infection to which in so many cases the infant is exposed at once, and it becomes easily intelligible why the children of tubercular parents so often become tubercular.

But although the knowledge that tuberculosis is infectious has, in a manner, added to its terrors, yet it is evident that contagion lies more directly in our own hands. We know to what degree and in what manner tuberculosis is infectious, and it is our own fault if we do not guard against it. The terrible fate which seemed to hang over the members of tubercular families in past days, the fate from which they could not escape, which dogged their footsteps and sooner or later dropped upon its victim, is now happily abolished, to the great gain of suffering humanity.

If the predisposition to disease may seem to some a subject of depression, and almost as direct a menace as the belief in the direct hereditary power of disease, the brighter aspect of the matter should be kept in view. We cannot get away from our inheritance, but our inheritance probably includes a majority of healthy ancestors. If the inheritance of constitutional defects prevailed to the extent which some writers would have us believe, the human race would have died out long ago. There is, above all, a toughness of constitution and a power of adapting oneself to bad

conditions, which appears pre-eminently heritable, and is, no doubt, the fitness which has been inherited from those who have survived in the struggle for existence. In the class of superior social standing this toughness of constitution is not nearly as noticeable as among the very poor, nor does it probably exist to anything like the same extent. The power possessed by infants and older people in the slums of great cities to survive, in spite of every external disadvantage of bad housing, bad food, cold, repeated attacks of serious illness, and neglect of the ordinary laws of health, is a perpetual wonder to those who visit them. We should not hesitate to realise the fact that the perpetuation of badly tainted stock, vulnerable to many ills or to any special disease, lies in our own hands, and it is our incorrigible supineness, our habit of shifting the responsibility on to Providence, which prevents our taking more effective steps to check the evil.

Marriages of people of different social rank, even of one who is rich with one who cannot bring a sufficient sum of money to the joint exchequer, are daily prohibited, and the world approves such caution. Yet we look on complacently to see a man or woman mated with one coming from a stock unsound to the core, and we have not the courage to cry out at such inhumanity towards their offspring.

Especially is this the case where an inherited lack of nervous stability exists in the family. The increase of insanity and feeble-mindedness is a most disturbing fact to the minds of all thinking people, besides being a constant drain upon the purse of the unfortunate ratepayer, who has to support armies of pauper lunatics, imbeciles and feeble-minded, and is confronted with the fact that this burden is not waning, but has grown with each generation.

"In 1904 one person in 287 of the population was under care as insane; in 1914 the proportion has risen to one in 266; and since 1859 the ratio of insane to population has risen from 18·67 per 10,000 to 37·59, whilst the proportion of females to male patients is constantly rising. The expenditure on asylums last year was £3,347,263."

In olden times we hear of families possessed of devils, or duly venerated and feared as witches and sorcerers; we now take a more prosaic view of such eccentrics, and speak of them as members of a neuropathic stock. Insanity is described as "a disorder or disease of the nervous system which prevents the individual from reacting normally as a member of the society to which by birth and education he belongs." Unsoundness of mind is the incapacity in a minor degree of reacting normally to circumstances.

To the former belong those who are chronically or occasionally so incapable of managing their lives that they must be detained. To the latter belong the eccentric, the feeble-minded, and many criminals, out of harmony with their surroundings, incapable of exercising mature judgment, impulsive and generally neurotic.

Such people of nervous instability are apt to become insane when attacked by disease, or if called upon to meet some sudden strain upon their nervous energy.

Insanity, or a neuropathic hereditary tendency in a parent, tends to re-appear in his children, not always as insanity, but in one of the forms of nervous instability, such as epilepsy, chorea, eccentricity, moral obliquity, idiocy, uncontrollable fits of passion, or some other form of nervous disorder. It must, however, be kept in mind that, where insanity or any such nervous disorder is due to an accident which has injured the brain, or to malnutrition of the brain during development, it is not transmissible. Not only must the brain have adequate nourishment, it is masterful in demanding a lion's share of that which goes to nourish the body, and when it is starved, especially at the time when it is actively developing, there is great danger that such development may be permanently arrested. Accidental injury to the brain is not transmissible; it is only nervous disorders of germinal origin which are directly heritable, and in the absence of more definite knowledge we must conclude that such disorders are of germinal variation, a sport, which, like other sports, can be inherited.

It is often difficult to differentiate in such cases; malnutrition of the brain, or prolonged nervous strain in the parent, must interfere with the nutrition of the germ plasm; and by this means the nervous system of the offspring may be affected, even to the extent of causing a primary defect in the development of the nerve centres, a defect which may persist as a definite variation.

Insanity in the mother is more potent in affecting

her offspring than insanity in the father. In this, as in all diseases where heredity plays a part, consanguinity, or the marriage of near relations, is a question of importance. Mere consanguinity is not, as some maintain, ever a cause of insanity. But close inbreeding, often resorted to by the breeders of animals in fixing a desirable character, must greatly augment the danger of perpetuating any morbid idiosyncrasy in the stock. There is no reason whatever why the marriage of first cousins who come of healthy stock should be interfered with. In fact, where the common grandparent had some desirable character which has since remained recessive, it may re-appear as the result of the marriage of first cousins. But where a morbid strain is present in their mutual ancestry, the danger of perpetuating that condition is greatly increased.

In the Report of the Commissioners in Lunacy for 1913, heredity is stated to be the principal agent in the causes of insanity.

The Commissioners state:

"Foremost amongst the circumstances in the life history of one who becomes insane is that of an inherited tendency to mental and nervous instability. For if, to the figures given for an insane heredity, there could be added those of a family history of nervous affections and of inebriety, the proportion of cases in which such inheritance was noted would amount to from 30 to 40 per cent., the ratio being higher in the female than in the male sex. . . . In about 7 per cent. of all the cases of first attack, no other factor than this one of inheritance could be definitely assigned as the causal agency."

Heredity also goes far to explain, in the vast majority of cases, why an individual becomes insane when he is subjected to depressing influences which would not have this result in one coming from a healthy stock. Dr. Mott, pathologist to the London County Asylums, says: "A neuropathic heredity is the most important cause of insanity. . . . There are individuals, born of sound stock, whom no acquired conditions—for example, drink, poisons engendered within the body or taken from without, head injuries, emotional shock, distress, or even profound misery and destitution, separately or even all combined—can render insane. There are others, and these are generally from a neuropathic stock, whose mental equilibrium may be upset by any one of these exciting causes."

As advocates of eugenic principles we can have no doubt that the men or women who ought to marry and have large families are the people of sound capacity and energy, and the more children of this type that England can produce the better for the nation. But in this world of all sorts and conditions of men, we must put in a plea for the occasional abnormal man, even though we may sometimes question the wisdom of his propagating his kind.

Some forms of genius and eccentricity are certainly allied to insanity. "Great men," says Galton, "may be even indebted to touches of madness for their greatness; the idea by which they are haunted, and to whose pursuit they devote themselves, and by which they rise to eminence, having much in common with the monomania of insanity."

Genius will serve its purpose and justify its presence in the world by the mark it leaves behind; but such gifted individuals, if they come of a neuropathic stock, seldom pass on their more worthy characteristics, but only the degenerate tendencies. Think what a monotonous, grey world this would be if it were not for the passing radiance cast upon it by visionaries such as Muhammad, Joan of Arc, Emanuel Swedenborg or Charles Gordon. Yet their visions might now be termed hallucinations. And consider the great epileptics, Julius Cæsar, Savonarola, and perhaps Napoleon Buonaparte, whose very instability gave rise to their intense and magnetic personalities.

Genius is not infrequently met with in neurotic families, and a man can be a genius with occasional fits of insanity. Those who devote their lives to work in which the imagination plays an important part, such as artists, poets, and actors, are liable to periods of insanity, "the blaze of brilliancy being often followed by a gloom of nervous exhaustion."

Genius of this type is not, in the words of Longfellow, "Genius, which means the transcendant capacity of taking trouble." That requires as its foundation—character, stability, and a disciplined mind; the most desirable qualities which a man can pass on to his offspring. The genius who consults the mental specialist at intervals is the man of unstable nervous capacity, swayed by emotion and caprice, alternating between flashes of real power and periods of incredible weakness. "Little genius,

indeed if any," says Sir George Savage, "is met with in asylums. . . . The insane have no remarkable powers, either bodily or mental, they may disregard general opinion or may be so self-absorbed as to appear to be gifted with undue powers, but essentially all insanity is weakness."

Genius, therefore, although it is not always allied to madness, is apt to occur in the same stock; and in some very rare instances we are told that a man of unsound mind has been the father of a genius. Of this Prof. Pearson says: "I shall be told that men of genius have occasionally had an insane parentage. My answer to that is that the average number of insane offspring of an insane parentage is about 25 per cent., and that when we follow these families of insane up to 50 years of age, it is more nearly 40 per cent. Are we, for the sake of a genius once in a century, to inflict the suffering denoted by these percentages, not only on the individuals themselves, but on their friends and relatives, and what is more important, on the nation as a whole?"

The neuropathic disposition, which is very heritable, and which may be the first evidence of degeneracy of a stock, shows itself in many different ways, not only in such evident signs as insanity, epilepsy or hysteria.

A great deal has been written by the advocates of temperance to try and prove the hereditary transmission of alcoholism; and where a question has long been the subject of such a hot, and I may say intemperate debate, it is difficult to get any exact facts. The violent partisanship of one party somewhat blinds their opponents as to the value of the arguments brought forward, and each accuses the other of narrow-minded prejudice.

Does alcohol actually produce mental disease, and is such disease transmissible? Dr. Mott says definitely that, although "alcohol may be a powerful co-efficient, it is not of itself the main cause of insanity. The quantity of alcohol which may be consumed daily by a man of inherited sound mind without apparent harm may be sufficient to make a potential lunatic anti-social and certifiable."

This is proved by the fact that those who are admitted to lunatic asylums for alcoholic insanity do not show signs of prolonged abuse of alcohol. In general hospital practice, on the other hand, many cases are met with where patients show, by a condition of advanced disease of the liver and other organs, that they have drunk persistently for many years, but because they inherited a stable mental organisation they never showed signs of insanity. We must conclude, therefore, that alcoholic excess does not produce insanity unless there is, to begin with, an inherited nervous instability, which makes the individual peculiarly susceptible to the poison.

Extreme cases of alcoholism are associated with mental defect, and the advocates of temperance have interpreted this fact into the belief that alcohol was the cause of the defect, without investigating whether the mental defect was present before the alcoholism or was consequent on the alcoholism.

But researches carried out by Prof. Pearson and Miss Elderton prove that, where alcoholic mental degeneracy occurs, there is already present germinal defect, that the individual comes of a neuropathic stock, and the alcohol is only the liberating stimulus which sends him over the border line into insanity.

Now, how does this affect the question of heredity? Do the children of heavy and continuous drinkers inherit that vice, or inherit mental defect? The researches go far to prove that this is not the case. The man who is capable of chronic and heavy drinking has inherited a sound mental and physical constitution, and he transmits these desirable qualities to his children; although if he has previously so poisoned his system with alcohol as to interfere with the nourishment of the germ plasm, his offspring will be physically and mentally less vigorous than himself.

From statistics collected it appears that "no marked relation has been found between the intelligence, physique, or disease of the offspring of parental alcoholism. On the whole the balance turns as often in favour of the alcoholic as of the non-alcoholic parentage." The misery of the drunkard's home, the atmosphere of terror, privation and neglect which is too often present, besides the influence of example, re-impresses the same conditions on the offspring, and tends to produce the same failing in successive generations; but this is due to a failure in nurture, not necessarily in Nature.

August 30th, 1915.

CLINICAL JOTTINGS.

(*Continued from p.* 248.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

Delirium Tremens and Traumatic Delirium.

Delirium *e potu* may arise under two different conditions.

In the one the patient has been drinking hard right up to the onset of the attack—has, in fact, drunk himself into it. This is the common "delirium tremens." These attacks are quite compatible during the intervals with temperate or even abstemious habits.

In the other the patient is a constant soaker, never perhaps actually drunk, but also never really sober. He goes on as usual till some sudden shock occurs, such as an accident or an acute illness, and then immediately he becomes delirious. This is often called "traumatic delirium."

The general condition is the same in the two groups. There is in both the same active, busy delirium with hallucinations, due to the same cause, viz. alcoholic excess; but in all other respects the two conditions stand in strong contrast to one another.

In delirium tremens alcohol should be at once cut off, and no sedatives given. The patient should be fed freely, and in two or three days will be convalescent.

In traumatic delirium the patient would not have had the attack except for the accident or illness. Alcohol and sedatives will both be necessary, possibly in considerable amount. The prognosis is very grave, for most of these patients die.

Though both groups are commonly called by the same name, they should be sharply distinguished and it would be well if they had different names. To emphasise this difference I used to suggest that if one was called "D. T." (delirium tremens), the other should be called "T. D." (traumatic delirium).

In many cases a ready means of distinguishing between the two groups lies in the temperature. In "D. T." the temperature is usually not raised, or not much if at all, whereas in "T. D." the temperature may range high, reaching 103° F. or more. This gives a guiding rule which is useful. If in a case of alcoholic delirium the temperature be raised, it is probably not with "D. T." and its favourable prognosis that we have to deal, but with "T. D." and all its differences of treatment and attendant danger to life.

The Relation of Palpitation and Tachycardia to Cardiac Lesions.

It is often stated that long-continued palpitation may lead to cardiac disease, the assumption being that the heart was healthy prior to the palpitation, and would have remained healthy if it had not been for the palpitation.

In support of this view the association of mitral regurgitation with exophthalmic goitre is the stock instance. This association is robbed of all its value by the frequency with which a history of rheumatic fever is obtained in cases of exophthalmic goitre, a frequency which amounts to 10 or 12 per cent. at least, while, on the other hand, it must not be forgotten that in at least 80 per. cent. of the cases of exophthalmic goitre, the palpitation may last for years without any sign of organic heart-lesion developing.

Tachycardia is not now so often quoted in support of the statement, for though this affection was orginally described as occurring in healthy persons, the general opinion now is that it is itself the result of serious organic disease of the heart, not perhaps obvious for a time, but becoming manifest later.

(*To be continued.*)

The Support of an Artificial Leg.—Suchier has worn an artificial leg for twelve years, and relates a number of points he has learned from this experience. The skin in and around the inguinal region is exceptionally tender and sweats readily, so that it gets sore easily. No pressure should be allowed on the skin in this region. The weight should be supported by the tuberosity of the ischium, just as the acetabulum bears the weight on the femur in normal conditions. The tuberosity of the ischium naturally bears the weight of the body when sitting, and it should be utilised as the support for the artificial limb, rather than the sloping surface of any bone.—*Journ. A.M.A.*, vol. lxv, p. 288.

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We regret that owing to the war the number of pages must be diminished for a time.

CLINICAL NOTES ON CEREBRO-SPINAL MENINGITIS.*

By J. RUPERT COLLINS, M.A., M.D.Dub. Univ., M.R.C.P.Lond.,

Hon. Physician Cheltenham General Hospital.

IT is my intention to present a short sketch of the leading features of cerebro-spinal meningitis, and then to point out some of the pitfalls associated with the diagnosis from a consideration of the cases I have seen. The disease is due to an infection with the diplococcus intracellularis of Weichselbaum, an organism characterised by being Gram negative, pleomorphic, growing freely on serum or ascitic agar, and of low resistance. This diplococcus sets up a meningitis affecting especially the spinal cord and the base of the brain. As a result, the spinal symptoms and affections of the cranial nerves are much more pronounced than the cerebral. The knee-jerks and Babinski's plantar reflex may be present or absent. Kernig's sign I have always found present. It is elicited by bending the thigh on the abdomen and then trying to straighten the leg on the thigh. When the sign is positive the leg cannot be extended beyond a right angle with the flexed thigh without evoking pain. Brudzinski's neck and leg signs are most valuable; the former is elicited by flexing the arms and legs and then placing the hand behind the patient's head and trying to bend the head forward on the chest. It will be found when the test is positive that firm pressure will not bend forward the head, but will simply lift up the patient's head and shoulders with extended neck. The leg sign is elicited by lifting up one leg as the patient lies on his back, when the other leg will be noted to raise itself up spontaneously. General hyperæsthesia of the skin is very noticeable, and a well-marked *tache cérébrale* is easily produced.

* Clinical Lecture at the Cheltenham General Hospital.

The most frequent mental condition is a heavy stupor from which the patient can be roused for a few minutes by shouting, but quickly relapses again.

Other patients show perfectly clear mental action, while still others are quite unconscious. The decubitus assumed by well-developed cases of cerebro-spinal meningitis is very characteristic. The patient lies on one side with his head retracted, his arms and legs flexed; in fact, very much the attitude assumed when climbing a rope, or what might be termed a monkey-up-a-stick attitude. Any attempt to alter this position is at once resented by the patient. The skin rashes are very variable, the most frequent in the cases I have seen is a petechial rash rather like flea-bites with a pressure erythema over the buttocks and knees. Most of the cases I have met with had no rash at all. Herpes round the mouth is frequently present.

The onset is usually sudden, but in some cases there is a preceding period of pharyngitis or malaise. The pulse is soft and not markedly altered in frequency, at times irregular. The temperature is not in any way characteristic, and may be normal or hyperpyrexic. In most of my cases it was irregular between 98° and 101° F.

There is nearly always severe occipital headache.

The course of the disease is as variable as its symptoms; death may take place within twenty-four hours, or the disease may run a favourable course and the patient be convalescent in a week, or the disease may drag on with ups and downs for two or four months.

An evil feature of the disease are the legacies it may leave behind in the shape of deafness, blindness, paralysis, hydrocephalus, or mental weakness.

In regard to treatment, in the first place it is advisable to keep the patient quiet and shade the room, as there is frequently hypersensitiveness to sounds and acute photophobia. It is also most important to have as much fresh air as possible. Soldiers have been noted as peculiarly subject to this disease, more particularly in cold weather. It is difficult to secure adequate ventilation in the sleeping quarters of a large number of men, and more so in cold weather. In all the houses in which I saw a case of this disease there was a very distinct "frowst." With open-air methods the risk of infection to the nursing staff will also be lessened.

The next step in treatment is lumbar puncture, which also affords the only certain means of diagnosis. The relief afforded is marked, and frequently a patient who is quite unconscious will recover and become mentally clear in a few hours after rachicentesis. We may also presume that thereby an effete fluid is replaced by one containing fresh bacteriotropic substances. The puncture should be repeated as often as necessary, and at first may be done every day with advantage.

Flexner's serum has been used with much success, especially in the United States and in Belfast. The different makes of serum seem to vary a great deal in efficiency. The serum should be used in 20 to 30 c.c. doses injected into the subarachnoid space by lumbar puncture, taking care to inject a smaller amount of fluid than was withdrawn, and to warm it to body-heat prior to injecting. Vaccines I have used to a larger extent and with very beneficial effects in some cases. Atoxyl has been used by Low in Africa, and 1 per cent. lysol injected into the spinal canal has been advised. But I do not think either methods are used much. The technique of a lumbar puncture is simple. The skin is sterilised, for which I prefer washing with chloroform and then placing a drop of pure lysol at a spot between the fourth and fifth lumbar vertebræ on a line drawn between the highest points of the iliac crests, and about half an inch from the middle line. The needle is then passed upwards and inwards. The more the back is arched the wider the space between the laminæ and consequently the easier to enter the canal. The puncture may be done under a local anæsthetic, but if the patient is restless or timid I prefer a general anæsthetic of which very little is required.

For insomnia, pain, and restlessness morphia is much the best drug.

In all probability, for some years after this epidemic is over we will have sporadic cases cropping up, and I will now quote a sporadic case I had ten years ago to show how it was precisely similar to the present cases.

A girl, æt. 13 years, complained of a chill on the evening of May 21st. The next morning she refused her breakfast and went to sleep. At twelve noon the same day her mother found her unconscious, having passed urine and fæces under her. I found her an hour later lying unconscious on her left side, her mouth wide open, both pupils small and equal in size.

The next day she was partially conscious, and complained of severe headache; herpes appeared on the upper lip and some livid coloured spots on the body. Kernig's sign was now present. A diagnosis of cerebro-spinal meningitis was made, and on the 25th I performed lumbar puncture.

Immediately on passing the needle into the spinal canal, a purulent fluid welled up, about 1 c.c. coming away. The pus cells were almost entirely polymorphs, and Weichselbaum's meningococcus was found and cultivated. The patient made a good recovery and still enjoys good health.

A boy, æt. 9 years, quite well previously, on January 12th of this year, complained of pain in his head and vomited twice. When Dr. Dighton saw him a few hours later he was unconscious, restless, and with a temperature of 105°. I saw him the next day with Dr. Dighton, and he was then in much the same condition. He was lying in the typical "monkey-up-a-stick" attitude, with purpuric spots distributed over the trunk and limbs. Kernig's and Brudzinski's signs were present. Lumbar puncture yielded a turbid fluid which, on examination, was found to contain pus cells and Weichselbaum's meningococcus. The child was treated with vaccine and serum, and on the sixth day of his illness looked as if he was going to recover, but on the next day he became rapidly worse and died.

The next case was a girl, æt. 3 years, who was sent into hospital by Dr. Richard Davies with the the diagnosis of cerebro-spinal meningitis. She had for three days been ill with attacks of vomiting and delirium. There was a petechial rash on the buttocks, and the child was very irritable. Kernig's sign was absent, and there was at first on admission no head retraction. Lumbar puncture yielded a fluid rich in pus and gave a good growth of meningococci. The patient was treated with vaccines and made a good recovery. It is important to note that it was not until the fourth day of the illness that Kernig's sign and stiffening of the neck muscles occurred.

The next case is of interest from its tragic suddenness and from the history, which might have been misleading. A girl, C. C—, became suddenly unconscious on April 4th, at 10 a.m. She had been apparently perfectly well and running messages half an hour earlier. She had had a slight discharge from the right ear for ten days, and this raised the possibility of a cerebral abscess. When Dr. Allman Powell saw her that morning she had vomited, and the back muscles were stiff. The child was sent into hospital the same day. A measly rash was present, most marked on the back of the thighs and buttocks. None on the body or face with the exception of a few urticarial spots on the abdomen.

Kernig's and Brudzinski's signs were present. A lumbar puncture yielded a fluid containing numerous pus cells and meningococci. The child died twenty-seven and a half hours after the onset of the illness.

The following case, admitted to hospital under the care of my colleague, Dr. Carter, shows how puzzling the early history of these cases may be.

A woman, Mrs. S—, at the end of February developed a cold, on account of which she was in bed for fourteen days. Prior to this she had been visited by her husband, who was in the navy, and who had just come out of hospital, having been treated for a sore throat and influenza. On March 12th her temperature rose to 105°, and she had diarrhœa. In four days her temperature fell to normal, but she continued to have headache and pain in the back. On March 19th she was delirious, and was admitted to hospital on the 20th. She was then very drowsy and had difficulty in understanding what was said to her. Her pupils were both widely dilated. Kernig's sign was present. The cerebro-spinal fluid was found to contain pus and meningococci. The patient has been treated with vaccines and had one injection of serum and numerous lumbar punctures. She is to-day, May 8th, better but mentally very dull.

A publican had an attack of erysipelas from which he apparently got well, but a fortnight later developed symptoms resembling delirium tremens. As the case seemed peculiar, Dr. Dighton asked me to see him. There was no rash, but both Kernig and Brudzinski's signs were present. The cerebro-spinal fluid was quite purulent and contained some flocculent masses. He was removed to the Delancey Hospital and has been treated with vaccines. He has been very ill, and when apparently well had a relapse, but Dr. Lloyd Davies, who had charge of him, tells me that he has made an excellent recovery.

On April 22nd Dr. Pruen asked me to see a girl, æt. 20 years, and make a lumbar puncture to

determine the diagnosis as to cerebro-spinal meningitis. She had a slight sore throat for three weeks previously, and her temperature during that period had varied irregularly round 101°. She had occasional attacks of vomiting and had severe pain alternating between the occiput and the sacrum. In the second week of her illness a very severe eruption broke out on her face. When I saw her, beyond the remains of the herpes there was no rash. Kernig's sign was present, and the neck was stiff. The girl was quite clear mentally. The cerebro-spinal fluid was only slightly turbid, but on examination was found to contain pus cells and meningococci. She was removed to the Dalancey Hospital under the care of Dr. Lloyd Davies. For some time after admission she was considerably worse. I performed rachicentesis again with apparently much benefit. She was treated with vaccines, and though at one time her condition seemed hopeless, she has made a good recovery.

A soldier was admitted into a V.A.D. Hospital with the history of pains in the head for five days, a filthy tongue, and very offensive breath. For fourteen days his symptoms were mainly those of gastritis. Nystagmus was now noticed, and on further investigation he was found to have Kernig's sign and rigidity of the neck. Double optic neuritis was present. His sister stated that he had three similar "head attacks" in the previous fourteen months. The question arose, was this a case of cerebral tumour or cerebellar abscess or of epidemic meningitis? Lumbar puncture settled the diagnosis, the meningococci being present. He was removed to the Delaney Hospital. He resolutely refused to allow a lumbar puncture to be done again and died after several weeks' illness.

A lady travelled from Plymouth to Cheltenham on March 27th, and the next day saw Dr. Hebblethwaite on account of a sore throat. The tonsils were inflamed. Her temperature became normal, her throat got better, and she seemed to be getting quite well. On April 5th, after lunch, she shivered, felt very ill, vomited and had a severe headache. She went to bed feeling very collapsed, and attributed the symptoms to something she had eaten. On the 7th the metacarpal joints of her left hand were inflamed, she had previously had rheumatic fever. On the evening of the 8th head retraction came on, and Dr. Hebblethwaite regarded the case as one of epidemic meningitis, and asked me to see her on the morning of the 9th. The patient was then quite clear mentally, the head was held retracted, and Kernig's and Brudzinski's signs were present. There was acute general hyperæsthesia, photophobia, and pressure erythema. The left metacarpal joints were swollen and tender. Lumbar puncture yielded a fluid containing pus and meningococci. The patient expressed herself as feeling much better after the rachicentesis, and the prognosis seemed relatively favourable considering the subacute onset, but she became suddenly worse and died at 3 a.m. on the 10th. This is the only case in this epidemic in which I found arthritis, a not unusual complication of the disease.

Mrs. B— had a discharge from her left ear for many years. During the three weeks previous to her admission to hospital she complained of pain in this ear; she also said that the back of her neck was stiff, which she attributed to keeping her head for a long time in one position on account of her ear trouble. She was admitted into the aural department of the hospital under Dr. Pike. There was no optic neuritis; there was slight spontaneous nystagmus, which later became more marked. There was a slight Kernig's reaction. No rash was present. The history looked very like an intracranial abscess. Dr. Pike asked me to see the case, and lumbar puncture yielded a cloudy fluid containing pus and meningococci. She became worse, had incontinence of urine and fæces, and died on the seventh day after her admission.

A post-mortem examination was made the day after death. Thick pus was found over the base of the brain; the pons, medulla, and the upper and lower surfaces of the cerebellum being most affected.

In the present year three cases have been sent to the Cheltenham General Hospital as suffering from epidemic cerebro-spinal meningitis which proved to be cases of tuberculous meningitis. Dr. Chalmers states that in Glasgow in 1908, when the epidemic there was subsiding, the proportion of cases notified as cerebro-spinal meningitis which proved to be tuberculous reached 16 per cent. The tuberculous form is so familiar that I will not give details of any case, but the pneumococcic meningitis is much rarer, and I will quote two cases to show how closely they resemble clinically those due to the meningococci.

On September 29th, 1910, I saw Miss V. A—, æt. 16 years, with Dr. Dighton. She had been to school the previous day, and came home complaining of headache. Her temperature rose the same night to 105° F. When I saw her twenty-four hours after the commencement of her illness, she was lying on her back, semiconscious. Some purpuric blotches were present on the right wrist, the knee-jerks were absent, Kernig's and Babinski's signs were both present. Lumbar puncture yielded a fluid only slightly opalescent, which was found to contain pus cells and pneumococci. The patient became quite clear mentally after the rachicentesis, and remained so during October 1st and 2nd, but on the 3rd she became unconscious. A lumbar puncture did not relieve her, and she died the same day, the sixth of her illness.

A. L. S—, a young girl, æt. 15 years, appeared to be in robust health up to Thursday morning, April 2nd, 1908, when she complained of pains in her head. At midday she ate some dinner, and then, not feeling so well, went to bed. About an hour later her mother, on going to see her, found her delirious, and sent for Dr. Blakeney, who saw her at 3.30 p.m. She was then in a convulsion and quite unconscious.

At 5.30 p m. I saw the patient with Dr. Blakeney, and found her moribund; superficial and deep reflexes absent. We agreed in the opinion that it was some form of cerebro-spinal meningitis, and arranged to meet again at 7 p.m. to perform a lumbar puncture. On our arrival at the house we were informed that the girl had died at 6.30 p.m.

I made a lumbar puncture on the dead body, and obtained a slightly turbid cerebro-spinal fluid, which, when centrifugalised, yielded a deposit consisting almost entirely of polynuclear leucocytes, and containing numerous pneumococci.

A post-mortem examination was made the following morning, and purulent meningitis was found on the convexity of the brain and more marked at the base. Pneumococci were found in the pus.

A consideration of these cases will show how difficult is the diagnosis very often in the early stages of the disease, and how the history may even be misleading. Symptoms of meningitis, if preceded by a sore throat, are very suspicious, and if a rash is present still more so. The diagnosis has to be made from other forms of meningitis, cerebral abscess or tumour, uræmia, or any other form of general septicæmia. The following are broadly the information afforded by examination of the cerebro-spinal fluid.

For lumbar puncture it is the sub-arachnoid space which is tapped, except in cases of meningitis serosa, when a fluid effusion separates the dura and the arachnoid, and the subdural fluid is then drawn off.

Acute inflammation of the meninges is most commonly caused by the pneumococcus, Weichselbaum's diplococcus, streptococcus, staphylococcus, Friedländer's bacillus, and the tubercle bacillus.

Sub-acute and chronic meningitis is usually due to the tubercle bacillus or to syphilis, and is characterised by a smaller exudate of leucocytes, of which the mononuclears form the larger proportion.

When a meningitis with a polymorph leucocytosis is recovering, the character of the cellular exudate becomes altered, and the polymorph yields place to the mononuclear cell. It is frequently very helpful to supplement an examination of the cerebro-spinal fluid by making at the same time a leucocyte count of the blood.

Roughly speaking, the indications yielded would, in most instances, be as follows:

A cerebral abscess will yield a high leucocytosis in the blood; and if no meningitis be present no leucocytosis in the cerebro-spinal fluid.

Meningitis due to pyogenic bacteria will yield a high leucocyte count in the blood and a polymorph leucocytosis in the cerebro-spinal fluid.

Tuberculous meningitis will yield a lower leucocyte count in the blood than that due to pyogenic organisms, and, as a rule, a mononuclear leucocytosis in the cerebro-spinal fluid.

A cerebral tumour will only in some cases give rise to a leucocytosis of the blood. Some cases of a cerebral glioma have had mononuclear leucocytosis of the cerebro-spinal fluid.

Syphilitic nervous affections, general paralysis, and tabes dorsalis give rise to a mononuclear leucocytosis. This also has been found in other affections, epilepsy and acute mania, by Dr. Williamson.

September 6th, 1915.

FOUR GRESHAM LECTURES ON HEREDITY.

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

LECTURE IV.

The Need for a Better Understanding of Eugenic Principles.

Atavism or reversion is a term applied to the frequent appearance of ancestral, but not parental, characteristics in an animal or plant. It is often said that a child takes after his grandfather or some other ancestor, and our national enemy, the Kaiser of Germany, supplies us with an interesting illustration of throwing back to a former, almost forgotten, ancestor. His peculiar temperament was certainly not inherited only from his heroic father, nor from his able, but sometimes tactless, mother.

It is hardly necessary to recall the insanity of his great-uncle, Frederic William IV (1840–1861), during the last three years of his life, but if we go back six direct generations, we find that exactly 200 years ago Frederic William I was reigning as the second King of Prussia (1713–1740). Macaulay, in his Essay on "Frederic the Great and his Times," has given us in a few words the unenviable character of the occupant of the Prussian throne in 1715. Frederic William I had some talents for administration, but his "eccentricities were such as had never before been seen out of a mad-house."

He was the first to form the design of obtaining for Prussia, chiefly by means of a strong army, a high place among the European Powers. His ambition was to form a brigade of giants, and every country was ransacked by his agents for men above the ordinary stature.

"These researches were not confined to Europe. No head that towered above the crowd in the bazaars of Aleppo, of Cairo, or of Surat could escape the crimps of Frederic William. One Irishman more than seven feet high, who was picked up in London by the Prussian Ambassador, received a bounty of near thirteen hundred pounds sterling—very much more than the Ambassador's salary. This extravagance was the more absurd because a stout youth of five feet eight, who might have been procured for a few dollars, would in all probability have been a much more valuable soldier."

The King's dominant passion was the love of military display, and the habit of exercising arbitrary power made him brutally savage. If His Majesty "met a lady in the street he gave her a kick and told her to go home and mind her brats. If he saw a clergyman staring at the soldiers, he admonished the reverend gentleman to betake himself to study and prayer, and enforced this pious advice by a sound caning, administered on the spot."

He despised literature, and thought that the only business in life was to drill and to be drilled. When his son was in his 'teens he was kicked and cudgelled; "at dinner the plates were hurled at his head; sometimes he was restricted to bread and water; sometimes he was forced to swallow food so nauseous that he could not keep it on his stomach. Once his father knocked him down, dragged him along the floor to a window, and was with difficulty prevented from strangling him with the cord of the curtain." The Queen and Princess Wilhelmina, for the crime of not wishing to see the youth murdered, were subjected to the grossest indignities. This warped education of the young man, known later as Frederic the Great, was, may be, responsible for many of that King's eccentricities, one of which was a love of severe practical joking. As Macaulay says of him, he showed "indications, not to be mistaken, of a nature to which the sight of human suffering and human degradation is an agreeable excitement."

In the last three lectures, we have seen some of the results brought about, first, by interference with Nature's methods of race improvement, which are the methods of natural selection, and of killing off the weak and worthless before they reproduce their kind; secondly, by our want of foresight in checking Nature's methods without substituting others to bring about the same result for the betterment of the race.

After slow and steady race improvement in the past, we are now artificially producing distinct race deterioration, and this is chiefly due to the obstinate belief that improved external conditions of life can transform the inborn qualities of man. The vast part which heredity plays in the fashioning of a race is scarcely considered by some philanthropists, who spend their energies in trying to better the surroundings of man, by education and by charity.

We have but to recall the brutal indifference of

our ancestors—whose methods of dealing with vagrants and unemployed was to hang them, forty at a time, on the gallows—to realise the tremendous swing of the pendulum which has brought us now to a condition approaching morbid sentimentality, when the most indigent and worthless is supported, from the cradle to the grave, at the expense of the thrifty, and his right to produce innumerable reproductions of himself is not questioned.

It stands to reason that the betterment of social conditions, which gives every individual a chance to prove his worth, must continue unabated. But that other greater factor, heredity, must in future be reckoned with as a chief power for racial improvement, a factor which must be taken into proper account in dealing with the health and the future greatness of a nation.

In this country there is but one driving power which compels our lawgivers to act, or bids them stay their hand when they are moving too impulsively; and that driving power is the will of the people. "Whenever public opinion is strongly roused," says Galton, "it will lead to action, however contradictory it may be to previous custom and sentiment. Considering that public opinion is guided by the sense of what best serves the interest of society as a whole, it is reasonable to expect that it will be strongly exerted in favour of eugenics, when a sufficiency of evidence shall have been collected to make the truths on which it rests plain to all. That moment has not yet arrived. Enough is already known, to those who have studied the question, to leave no doubt in their minds about the general result, but not enough is quantitatively known to justify legislation or other action, except in extreme cases. Continued studies will be required for some time to come; and the pace must not be hurried. When the desired fulness of information shall have been acquired, then, and not till then, will be the fit moment to proclaim a Jehad, or holy war, against customs and prejudices that impair the physical or moral qualities of our race."

It is public opinion which must be educated and which, moving discreetly, must insist on rational laws which do not make life too easy for the wastrel, nor check the increase of better stock, nor allow this country to become the rubbish heap on which all the worst elements, physical and moral, of Europe are cast.

Although the extent of our knowledge is not ripe for any positive assertions or dogmatic teaching, there are many ways in which popular opinion can work very effectively. It should create an atmosphere of social opinion which should operate powerfully against these dangerous conditions. England should ever stand for liberty, but we must beware lest, in the words of Milton, "License they mean when they cry liberty."

The placing of the Mental Deficiency Bill on our Statute books has been a great step in the right direction, though even here the voice of certain sentimentalists deprived it of much of its value. Many suggestions are made for active legislative interference which should tend towards eugenic reform, but in most cases we are reminded of the old saying that "Fools rush in where angels fear to tread." The more we know, the less we are inclined to interfere by drastic methods or hasty popularisation, both of which tend to bring the whole subject into ridicule, with the resulting danger that it will loosen its hold on the imagination of the thinking public.

In certain States of America various laws have been passed for the sterilisation of the unfit, and for the restriction of marriage licences to those only who can show themselves free from communicable disease, or who can produce a health certificate. But such laws, perhaps admirable in theory, have been passed impetuously to satisfy the enthusiasm of a few, and because they are premature they are already ceasing to be enforced. It is the old story that legislation is ineffective unless it has the strength of popular opinion behind it. But if the principles of eugenic reform can sink into the heart of the people, as assuredly they will when they are better understood, there will arise an enthusiasm for the cause and it will become such an ideal to improve humanity that, even if State action be not demanded, that strongest of all forces, the sense of the ordinary man and woman, will bring about what acts and statutes never could achieve without it.

Then at last we may look for such improvements as the permanent segregation of undesirables and deficients, while the State will be forced to diminish the burden of rates and taxes for prudent parents and will give other justifiable relief to encourage the best among young people to embark on matrimony.

It has been argued that eugenists desire to create a world of monotonously good people, all healthy in body and mind, stereotyped and eminently worthy. This would indeed be a sorry result! But, though many of us fortunately differ in our definition of the best type, we must all agree that strength of character, energy, intellect and bodily health are qualities which make an individual a desirable citizen; and the opposite qualities of a weak will, idleness, foolishness, and ill-health are qualities which ought to be eliminated from our race.

Never before has this question been of more momentous importance to us than now, for we have to face a crisis in our national existence which has made us ask ourselves whether England still possesses the tenacity, the perseverance and determination, both of men and women in all ranks of life, which characterised the Britons of old and which is needed to carry on the life and death struggle in which we are engaged. Time and the great issues at stake alone can answer the question, but we may be thankful that England has never yet failed to give an instant and enthusiastic response where enterprise and danger call, be it on the battlefield, on ocean, or the air.

And though the toll she pays is heavy, and many of the best of her young lives have been sacrificed, their places are still filled by others ready to take up the burden of her responsibilities.

It is of vital importance that the ranks of her future citizens should be filled with the able in body and mind, and though we deplore the ravages made by war, which robs us of the flower of our manhood, we know from the experience of these memorable months that it calls forth the best that is in us, the love of our country and the determination to defend her honour, which is stronger than the mere love of safety and worldly ambition.

If our young men had no opportunities of showing their mettle, their very courage might wither for want of use.

To no nation is a high type of manhood more necessary than to our own; for our race peoples the far corners of the earth, our traditions and our codes form the basis of human morality in every quarter of the globe, and we must beware lest any but the best in body and mind be the bearers of this splendid heritage.

"Go from the East to the West, as the sun and the stars direct thee,
Go with the girdle of man, go and encompass the earth.
Not for the gain of the gold; the getting, the hoarding, the having,
But for the joy of the deed; but for the duty to do.
Go with the spiritual life, the higher volition and action,
With the great girdle of God, go and encompass the Earth."

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We regret that owing to the war the number of pages must be diminished for a time.

THE DIAGNOSIS OF PNEUMONIA.*

By JAMES ALEXANDER LINDSAY, M.D., F.R.C.P.,

Professor of Medicine in the Queen's University of Belfast, Physician to the Royal Victoria Hospital.

IN a typical case no disease is more readily recognised than pneumonia. The sudden onset in the midst of perfect health, the initial rigor, the rapid rise of temperature, the marked dyspnœa, the thoracic pain, the characteristic sputum, the early prostration, the labial herpes, the flushed cheeks, the movements of the alæ nasi, constitute a clinical picture which admits of only one interpretation. Yet, mistakes in diagnosis are frequent in the early stage, and not unknown even in the later stages of the disease. It is not unprofitable to inquire how such mistakes arise, on what they depend, and how they may be guarded against. We might classify the causes of error somewhat as follows:

(A) Mistakes which arise from the misleading character of the initial symptoms.

(B) Mistakes which arise from the obscurity of the pulmonary signs.

(C) Mistakes which arise from the different manifestations of the disease in children, adults, and the aged.

(D) Mistakes which arise from peculiarities in the case depending upon the patient's constitution, history, and habits.

(A) The initial symptoms, though tolerably uniform upon the whole, may present wide variations. The onset may not be absolutely sudden, but may extend over 2, 3 or more days. Such cases are often labelled "Influenza, followed by pneumonia," but are probably in many cases pneumococcal from the start. The initial rigor is often absent, especially in children and the aged. The

* A Clinical Lecture delivered at the Royal Victoria Hospital, Belfast.

first symptoms may be mainly nervous, viz., intense headache, delirium, convulsions, even retraction of the head and strabismus, and the unwary practitioner makes a premature diagnosis of meningitis. This error is easily guarded against. A glance at the freely working alæ nasi; an examination of the chest; due attention to the temperature and the suddenness of the onset will prevent mistakes. Meningitis always sets in with a few days of vague malaise, restlessness, and hyperæsthesia. The temperature is rarely high, the chest will probably be clear, and the pulse and the abdomen may give useful hints. This mode of onset, *i. e.* an onset with symptoms of "meningism," is relatively common in the child and rare in the adult.

In another type of case the early symptoms are gastric, or abdominal, viz. vomiting, pain in the epigastrium, the right hypochondrium, or the right iliac fossa, and occasionally jaundice. This abdominal syndrome has sometimes led to the most disastrous errors in diagnosis: Appendicitis, perforation of the stomach, cholecystitis, even acute pancreatitis has sometimes been diagnosed, and unnecessary operative measures undertaken. Such mistakes will not occur if the practitioner takes a comprehensive view of his case. Due attention to the mode of onset, the dyspnœa, the sputum, the state of the chest, the temperature, the patient's aspect, and the course of the case will prevent error. It should be a cardinal rule always to explore the chest thoroughly in every case of sudden illness attended by abdominal pain and sharp pyrexia.

(B) The pulmonary signs of pneumonia are often latent for 1, 2, or 3, rarely for 4 days. The mode of onset and the general symptoms may make the diagnosis practically certain, yet the most careful exploration of the chest may yield only negative results. This has been my experience in quite a considerable number of cases, the explanation probably being that the pneumonia has begun centrally. Such cases are trying to the practitioner, who may be morally certain of his diagnosis, but obliged to confess to anxious friends that he cannot confirm it by the usual methods of examination. A waiting policy is indicated, as a few hours, or at most a day or two, will make the great majority of these cases clear. Before the practitioner pronounces the pulmonary signs negative he should make sure that he is not overlooking a limited consolidation of the apex. This is a not unusual source of error, especially in children. In some cases the early signs are not the typical pneumonic crepitus, followed by blowing or bronchial breathing, but simply enfeeblement of the vesicular murmur, leading sometimes to an erroneous diagnosis of pleural effusion. This error can be guarded against by attention to the mode of onset, which in pleurisy is rarely of the fulminating type so common in pneumonia, by inquiring for the sputum, and by an investigation of the cardiac impulse and the general physical signs. Diminution of the vocal fremitus, so characteristic of effusion, is not unknown in commencing consolidation. In pleurisy the physical prostration and the general toxæmic symptoms are usually much less marked than in pneumonia. If necessary, exploration may be resorted to, but is not often required at this stage. The condition of the urine may give important aid. In pneumonia a diminution of chlorides is the rule, and albuminuria is common —features improbable in pleurisy.

(C) One of the most frequent sources of error in the diagnosis of pneumonia is forgetfulness of the fact that the disease presents points of marked contrast in the child, the adult, and the elderly. Pneumonia in the young child often exhibits the following features:

(*a*) The onset is often characterised by the absence of an initial rigor, and by the presence of headache, vomiting, convulsions, and often by "meningism."

(*b*) The course of the temperature is often irregular.

(*c*) Sputum is usually absent.

(*d*) Pleuritic pain is often slight.

(*e*) The physical signs in the chest are often obscure.

(*f*) The subjective sense of dyspnœa is less than in the adult, while the respirations are often more frequent.

(*g*) The course of the case is often irregular and untypical.

In the elderly, pneumonia is often more or less latent, *i. e.* the symptoms are ill-marked and uncharacteristic, and diagnosis is effected only by a thorough physical examination. In all obscure illness in the aged the possibility of pneumonia should always be borne in mind. In such cases

the temperature may be only slightly raised and dyspnœa may hardly be sufficient to attract attention, but the physical signs are usually decisive.

(D) Pneumonia may present aberrant features depending on the patient's constitution, habits, and history. In the alcoholic we may find in one type symptoms of intense cerebral excitement, leading sometimes to an erroneous diagnosis of acute mania. These patients have sometimes been haled off incontinently to an asylum, where the error in diagnosis is speedily recognised, to the great injury of the practitioner's reputation. In another alcoholic type early and marked circulatory weakness is the leading feature. In a third alcoholic type pyrexia may be slight or even absent. This last type is very misleading, and care is required to avoid error.

In general, the diagnosis of pneumonia will not be often missed if the practitioner once definitely suspects the presence of the disease. The danger is that owing to aberrant modes of onset, misleading symptoms, or negative signs the question of pneumonia may not seem to arise, some alternative diagnosis thrusting itself upon the attention. Perhaps we might formulate a few clinical rules as follows:

(1) In all cases of sudden illness attended by pyrexia and embarrassment of respiration, inquire carefully for pneumonia.

(2) In all cases of sudden illness attended by symptoms of "meningism," inquire carefully for pneumonia.

(3) In all cases of sudden illness attended by abdominal pain and pyrexia, do not forget, after a thorough exploration of the abdomen, to make an equally thorough exploration of the thorax.

(4) Do not forget that sudden illness attended by headache and vomiting may find its explanation in pneumonia.

(5) Recollect that in connection with the diagnosis of pneumonia special difficulties may arise in the case of children, the aged, and the alcoholic.

(6) In all cases of doubt consider carefully the mode of onset, the temperature, the state of the chest, the sputum, and the urine.

September 13th, 1915.

SURGICAL SHOCK.

By J. FRED LESSEL, M.R.C.S., L.R.C.P.,
Anæsthetist, Victoria General Hospital, Halifax, N.S., Canada.

WHEN a patient is found, after a severe injury or operation, to be in a state of depressed vitality with a cold skin, low temperature, rapid pulse of low blood-pressure, he is usually said to be in a state of shock. This condition is very common, but the series of changes in the circulation leading to it do not seem to have been fully followed and investigated during the course of the injury. From repeated observations on the facial artery during operation I have noted the following reactions, which have considerable importance in the diagnosis and treatment of shock.

(1) When sufficient irritation is applied to an area of the body, a decided contraction of all arterial muscle takes place.

(2) This contraction is sustained while the irritation is sustained, and if sufficiently severe the pulse will be obliterated. If the irritation is sufficiently severe and prolonged, the obliteration of the pulse will be permanent, and immediate death result.

(3) After removal of the stimulus the artery dilates partially, but incompletely, and does not regain its normal size for some time (Fig.).

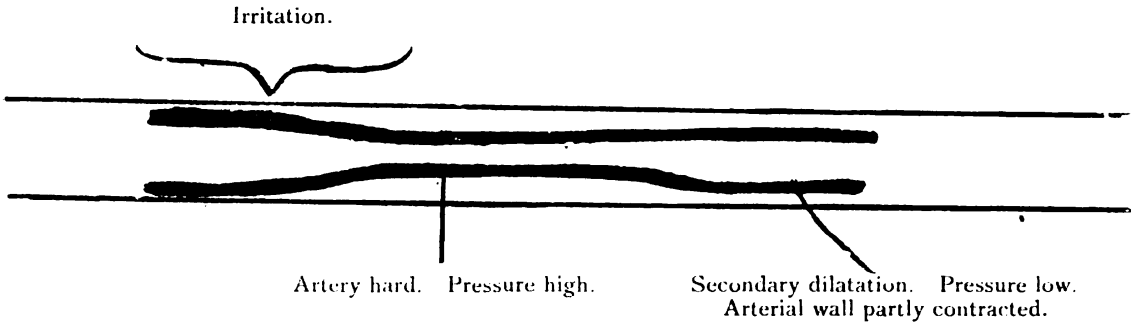
(4) With the initial irritation the pulse-rate does not rise, in some cases it may fall, but as soon as the irritation is removed, and the artery begins to dilate, the pulse-rate rises rapidly.

(5) Blood-pressure probably rises with the initial contraction, but falls rapidly with the secondary dilatation.

(6) Certain regions are especially susceptible to irritation, namely, the anterior parietal peritoneum and sub-peritoneal tissues in the lateral abdominal regions. Traction applied to any solid or hollow abdominal viscus will produce similar changes in the blood-pressure.

(7) Certain regions are relatively immune, namely, the groin, the brain, the sac of a hernia, the axilla, and the central line of the abdomen below umbilicus.

(8) The alternate application of irritation and its removal results in a small, hard, contracted



artery, with a low blood-pressure—the kind of pulse usually found after major operations, and usually termed the pulse of shock.

(9) These changes take place quite as regularly when the patient is under the influence of chloroform or ether as when fully conscious.

(10) The small hard pulse of low tension may be caused by mental impressions *only*, such as fear, pain, grief, worry, etc.

Shock then is a true reflex, and will be modified in a like manner. It may be brought about in three ways:

(i) Peripheral irritation carried by a direct path below the cortex to the vaso-motor centre. This may be termed peripheral shock.

(ii) Peripheral irritation carried to the cortical centres for the appreciation of pain and injury, and from these centres to the vaso-motor centre. This may be termed periphero-central or indirect central shock.

(iii) Impulses arising directly in the central nervous system, such as fear, worry, apprehension. This is direct central shock.

I. Cases illustrative of Peripheral Shock of Different Grades of Severity.

Case 1.—Operation: appendicectomy, no preliminary medication, deep ether anæsthesia, no reaction to skin, fascia or muscular division. The operator then elected to tear down on the peritoneum. Immediately there was a reaction—respiratory movements increased in depth, face became pale, veins of neck, chest, and arms engorged, facial artery contracted, became hard, and lost some of its tortuous curves, showing shortening. The irritation was only of a few seconds duration, and when removed the artery dilated to nearly normal, became soft, the face resumed colour, the veins subsided, and the pulse-rate rose from about 70 to 100.

This illustrates moderately severe transitory shock.

Case 2.—Intestinal obstruction. At the completion of the operation large masses of intestinal coils, which had been removed from the abdomen and which had been lying quietly protected by wet towels, were quickly swept into the abdomen. The facial artery contracted to a hard pulseless cord in four or five beats, remained so for twenty seconds, then resumed pulsation through a hard contracted artery to one of more rapid rate and of increasing softness and elasticity.

This illustrates severe transitory shock; no doubt if prolonged it would have caused immediate death.

Case 3.—Perforated duodenal ulcer thirty hours previously; general peritonitis. Pulse hard, wiry, contracted, rate 120. No alteration of character or rate under anæsthesia (ether). On opening the abdomen, intestines bulged sharply into the incision, due to enormous gas pressure in the bowel. Pulse became still more hard and small, and in three or four pulsations ceased to beat. A few spasmodic respiratory movements were made and all life ceased.

This illustrates very severe shock sustained and added to pre-existing shock which had not been abolished by inhalation anæsthesia.

Just here it might be well to note that the pulse usually described as the pulse of peritonitis—hard, wiry, "running" in character—is in reality the pulse of severe nervous irritation or the pulse of shock, whether that irritation be chemical, bacterial, toxic, or mechanical, in or out of the abdomen.

II. Indirect Central Shock.

Case 4.—Italian labourer unable to speak English. Severe lacerated injury to the leg received in a railway accident about twenty-four hours previous to admission to hospital. He was very much perturbed mentally and no doubt in much pain. Pulse rate 110, quite hard and contracted, but when placed under the influence of ether, pulse fell to 70 and became full and bounding. Amputation performed, and pulse did not subsequently rise above 80.

This illustrates shock due to mental impression combined with pain passing to the cortex and thence to the vaso-motor centre. When the cortex was cut off by the anæsthetic, shock vanished.

Numerous similar cases, as burns, superficial injuries, etc., readily illustrate this variety of shock.

III. Direct Central Shock.

Cases to illustrate minor degrees of this variety may be demonstrated in the wards of those about to undergo operation. The patients, usually men, will deny this fear, but acknowledge nervousness. The pulse will be found above 100, and a degree of contraction will be easily felt if one is accustomed to look for this sign. By reassurance or by means of a hypodermic of morphia and scopolamine this pulse may be made to resume a normal character.

Cases of exophthalmic goitre show this pulse frequently.

Grief, sudden bad news, has caused death. Pallor of the face, showing arterial contraction, is a well-known clinical sign. Joy, on the other hand, is characterised by a flushed face and has the reputation of never killing.

The act of vomiting is accompanied by pallor.

Excitement, as of a soldier in battle, shows peripheral dilatation and immediate shock is little felt.

Death has been caused by minor operations; and the use of the vaso-constrictor adrenalin on a mucous membrane seems to predispose to this event.

The statement has been made that shock is a reflex action and if reflex actions are abolished, shock is not present. This is true where concussion has preceded or accompanied injury.

Man, æt. 75 years, struck in the left side by a shunting engine with sufficient force to comminute all the bones of the arm into pieces about two inches in length. He was knocked down, and the wheels passed over his left foot—surely a very grave injury. On admission to ward, pulse was 40, weak, and hardly perceptible; skin cold, inelastic, and cadaverous; knee-jerk absent. In about two hours pulse rose to 80, full, and very soft. Patient next day stood amputation of both leg and arm without increase of pulse tension and without subsequent acceleration.

In this case the man complained of very little pain, and the general concussion of the whole body protected him against nerve irritation and shock—the artery showing absolutely no contraction.

Probably the most vexed question in the study of shock has been to explain a low blood-pressure with contracted arteries. Low blood-pressure could be explained on the assumption of an exhausted vaso-motor centre (Crile), but a contracted artery could not. The logical explanation seems to be that there is no exhaustion of the vaso-motor centre, that the contracted artery is contracted as regards normal size, but is really dilated relatively to the contracture which took place at the time the injury or nerve irritation was inflicted. This seems to fit all the known facts, and correlates the several schools of thought on the subject. The low blood-pressure will cause general arterial anæmia and form a vicious circle. Cardiac anæmia results in diminished cardiac contraction, and so little force is behind the blood in the aorta that it fails to overcome the contraction of the artery. This condition is usually termed collapse.

The distribution of the blood fluid in shock is of interest, and may be arrived at by a process of exclusion. It is not in the arteries, as they are contracted and blood-pressure is low. It is not in the surface veins, as these are collapsed. The fluid portion of the blood has not escaped from the vessels, as œdema is not a feature of shock; and Rendle Short has shown that the specific gravity of blood is not raised. There remain only the great veins of the splanchnic area, where, I believe, the great bulk of the vital fluid is regularly found. In cases of shock which come to post-mortem examination there is direct evidence of venous pooling.

Of the contributory factors in shock, hæmorrhage is probably the most profound; viewed in the light of the preceding arterial changes the loss of even one ounce of arterial blood makes a very grave impression on the already severe arterial anæmia.

Loss of body heat is probably second in importance. Coldness of the extremities—hands, feet and ears—are good diagnostic signs of shock during operation.

Acapnia in itself is not a direct cause of shock; in fact the great muscular relaxation present in this condition may protect the patient to a certain extent. But on the other hand, if acapnia develops after shock is established, the double condition would be extremely fatal.

Diagnosis.

Shock has always been a matter of opinion, there has been no certain sign to distinguish it from concussion, hæmorrhage, acapnia, heat exhaustion, etc. But if an estimate is made of the contraction of the arterial wall, shock may be diagnosed as either alone or complicated by other sources of lowered vitality. During operation the surgeon may be warned of causing nerve irritation, and the exact seats of susceptibility mapped out and avoided. By carefully following the artery during operation and using reasonable judgment, the pulse rate of a patient four hours after may be prophesied to within ten beats.

Treatment.

I shall discuss only a few of the best known and most frequently used measures and drugs, because my experience in the light of arterial constriction only covers the rather limited field of a few hundred cases.

(1) The anæsthetic. Inhalation anæsthetics, N_2O, chloroform, and ether, do not cut off peripheral shock unless carried to such a depth that blood-pressure is greatly lowered, in other words, unless fourth stage anæsthesia is established. This stage is rightly considered by most authorities to be too dangerous a degree. There are, however, occasional cases, such as Case 3, where arterial spasm must be overcome, so that we would be justified in giving a much deeper anæsthetic than would be allowable under ordinary circumstances.

Inhalation anæsthetics do, however, cut off direct and indirect central shock.

(2) Alcohol is an anæsthetic and vaso-dilator. A person so well under the influence of spirits as to be called drunk suffers very little shock from injury while in this condition.

It would be good practice, then, to administer alcohol immediately after injury where pain and arterial contraction are in evidence, but very bad practice to give alcohol where pain is absent and pressure low. Alcohol is thus contra-indicated in collapse following operation. The question to give or withhold alcohol may be answered by asking ourselves, would a light ether anæsthesia do our patient good? If yes, give spirits; if no, withhold.

(3) Strychnine increases the reaction of muscle to nervous influence. The value of strychnine in shock has been frequently discussed; it may be settled quickly. If shock is expected do not give it. If shock is present also withhold it. If shock has passed off, leaving effects which are sometimes known as collapse, strychnine may be of value. In all cases where arterial contraction is due to over-stimulation of the vaso-motor centre, strychnine is contra-indicated.

(4) Morphia depresses all reflex actions, both on vessels and on muscles. It is of exceptional value in shock expected and in shock present, but must be used carefully. No single dose should be over $\frac{1}{4}$ gr. and the dose should not be repeated more often than every fifteen minutes. It should not be given again after its effects are manifest for about four hours.

Morphia $\frac{1}{6}$ gr., combined with atropine $\frac{1}{150}$ gr., or scopolamine $\frac{1}{150}$ gr., should be given before all operations, whether under general, local, or spinal anæsthesia.

(5) Hyoscine hydrobromide or scopolamine in a dose of $\frac{1}{150}$ gr. combined with morphia $\frac{1}{6}$ gr. is an ideal preliminary medication dose. It dissociates the different centres from their action on the vaso-motor, prevents central shock and periphero-central shock by cutting out the memory for previous injury and putting the patient in the same class as a new-born baby with no previous impressions, the new-born being relatively free from shock.

(6) Saline infusion, subcutaneous, intra-venous, and rectal are all valuable, but the most valuable of these is rectal in massive amounts. The patient is elevated 18 in. at the feet, and saline solution at 115° F. is poured in the bowel until it will hold no more, from 40 oz. to 80 oz. or even more are used. It has three effects: (1) It exerts its direct pressure on the splanchnic venous area: (2) it introduces 5 lb. of fluid at a temperature 17° F. above that of the body; (3) it is directly absorbed. Rectal saline is quick, effective, easy to

give by inexperienced assistants and with little apparatus; also it does not interfere with intra-venous or subcutaneous injections.

Intra-venous infusion has the objection that the cases that need it the most are the most difficult to treat. It is useful where intra-rectal is contra-indicated as where a colotomy has been performed or where an anastomosis of the large bowel has been made, otherwise it should be preceded by intra-rectal 40 oz. to prevent blood dilution, by exerting pressure on splanchnic veins.

Subcutaneous infusion—under the breast in women, into the axilla in men—is useful where the previous two cannot be used, but is much inferior in its effects.

(7) A drug is required in the treatment of shock that directly increases the force of the heart without much effect on the vessels. Pituitary extract seems to have a greater effect on the heart than on the vessels. It is not ideal, but with its tonic effect on the bowel the general net result is to get the blood back out of the abdominal veins into the arteries. It is certainly valuable in doses of 1 c.c. every one, two, or three hours for three doses.

(8) Camphor is also a valuable cardiac muscular stimulant and should be used in 3 gr. doses hypodermically in oil every hour for five or six doses.

(9) Nitroglycerine should have a place in the treatment of arterial spasm. It should be used in doses of $\frac{1}{100}$ gr. hypodermically and repeated in ten minutes if its effects are not manifest. Its effects last about three-quarters of an hour.

(10) Heat to the surface is valuable in keeping up the peripheral circulation; it is of value in shock expected or present, or in collapse.

Routine Treatment.

(1) Shock absent but expected. Give morphia $\frac{1}{6}$ gr. in combination with atropine $\frac{1}{150}$ gr., or better scopolamine $\frac{1}{150}$ gr., one hour before operation.

During operation use Crile's anoci association methods by infiltrating skin and tissues with novocaine $\frac{1}{100}$. Especially remember that while cutting the peritoneum is almost shockless, any drag on parietal or visceral peritoneum is followed by immediate and intense shock. In cases of intestinal obstruction, the bowel may without shock be easily emptied by Moynihan's tube, provided gentleness in handling the intestines is carried out. This procedure is far less dangerous, even if the tube is inserted several times, than overhauling distended loops of bowel. Time is no objection provided there is no shock.

(2) Shock present and further shock expected. Everything possible must be done to unlock the arterial spasm present. Morphia $\frac{1}{4}$ gr. every fifteen minutes until it has its physiological action established. Chloroform in this case is the best anæsthetic, and the anæsthesia must be carried so deeply that the artery wall relaxes notably. This would be a very dangerous procedure in ordinary cases, but desperate diseases require desperate remedies.

(3) Actual nerve irritation practically absent but results of irritation present, such as are found in post-operative collapse. Elevate foot of bed, rectal saline 40-80 oz. at a temperature of 112°-115° F. Pituitary extract 1 c.c. every hour for three doses. Intra-venous saline 40 oz., camphor 3 gr. in oil every hour hypodermically for five or six doses. Possibly strychnine $\frac{1}{20}$ gr. every three hours. I have no doubt that an ideal drug will yet be found for the treatment of shock, when it is understood that a cardiac muscular stimulant is required that does not at the same time contract the arterial muscle.

September 13th, 1915.

Powdered Cinchona with Iodine for Infected Wounds.—Mouchet and Malbec recommend the following powder, which they name quinidol, as a substitute for iodoform in the dressing of infected wounds. It has a somewhat pleasant smell and costs less than a sixth of the price of iodoform, while its antiseptic power is at least equal to that substance. It is prepared as follows: For a powder to contain 5 per cent. of iodine, dissolve 5 gm. in 100 c.c. of ether and add 100 gm. of powdered red cinchona. Triturate the mass till complete evaporation of the ether has occurred. Then pass the powder through a fine sieve. A 10 per cent. powder requires double the proportion of iodine in a somewhat larger quantity of ether. The stronger powder is indicated in gangrenous wounds, the weaker in those which are atonic or only moderately infected.—*Paris Médical*, p. 307.

CLINICAL JOTTINGS.

(*Continued from p.* 288.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

Diabetic Retinitis.

There is much difference of opinion on the subject of diabetic retinitis. Some maintain that there is no such a thing; others that the retinitis which occurs is albuminuric and due to the association of diabetes with granular kidney; while others hold that there is a form which is perfectly characteristic and pathognomonic.

I have seen many cases of retinitis in diabetes, and I may say at once that I have failed to observe anything whatever characteristic in it—that is to say, nothing by which from the change in the retina diabetes could be diagnosed, as granular kidney can. I have had cases shown me more than once which were said to be typical, but they did not convince me, and so far as my own experience goes I should say that it was impossible to diagnose diabetes from anything that can be seen in the retina.

I agree, too, with the statement that many cases of so-called diabetic retinitis are typically albuminuric, and are due to the association of diabetes with granular kidney, an association which is by no means rare. But this does not exhaust the subject.

There are two forms of albuminuric retinitis which I have called the degenerative and the exudative respectively.

The degenerative form is the familiar and well-recognised type characterised by the white glistening patches of degeneration of varying sizes, and grouped especially round about the yellow spot, but whether large or small equally characteristic and quite pathognomonic, for they occur with no other disease.

In the exudative form there is exudation involving the disc and extending from the disc outwards in any direction; the patches of exudation may be of larger or smaller size, but if small never present the bright glistening appearance characteristic of the other form, but are of a dull yellowish colour, and a woolly or fluffy character, with no sharply defined margins. There is nothing in this form to distinguish it from the neuro retinitis of other affections—*e. g.* that which occurs with cerebral tumour. This form may occur in acute nephritis as well as chronic. It may disappear entirely as the nephritis recovers, while the degenerative patches are persistent and practically never disappear, probably because the renal lesions are permanent.

The exudative form is in all cases, I believe, of toxic origin, and might therefore arise in the course of diabetes, just as it does in the course of acute nephritis, and this, in my opinion, is the fact, for I have certainly seen cases of this kind in which there was no evidence of granular kidney.

The view I am expressing would reconcile and explain the different opinions held. The degenerative form would be characteristic of granular kidney, whether associated with diabetes or not. The exudative form would be of toxic origin and indistinguishable from that which is seen in other toxic states, and be due to some poison produced in diabetes which attacks the optic nerve and retina just as it sometimes does the peripheral nerves. What the poison is we know no more in diabetes than we do in nephritis.

(*To be continued.*)

Clinical Forms of Whooping Cough.—Shiperovich reports four cases of whooping cough with unusual clinical manifestations. In one there were acute emphysema of the lungs, emphysema of the entire skin of the body, head and extremities, and pneumothorax, the case terminating fatally from rupture of some alveoli in the lungs. In the second case the attacks of coughing were followed by unconsciousness; necropsy showed hæmorrhagic encephalitis and pulmonary œdema. In the third case the unconsciousness, clonic convulsions and glycosuria were probably caused by capillary hæmorrhage in the fourth ventricle. The glycosuria disappeared afterward, and the patient recovered. In the fourth case bloody tears showed during the cough. They contained numerous lymphocytes. This child also recovered.—*Journ. A.M.A.*, vol. lxv, p. 657.

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We regret that owing to the war the number of pages must be diminished for a time.

UNUSUAL MANIFESTATIONS OF POLIOMYELITIS.*

By F. E. BATTEN, M.D., F.R.C.P.,
Physician, Hospital for Sick Children, Great Ormond Street; and Physician to Out-patients, National Hospital for Paralysis and Epilepsy, London.

THE following series of cases illustrate some of the unusual manifestations of poliomyelitis. The word "poliomyelitis" is used in its widest sense to indicate an acute specific fever which may affect any part of the nervous system. Recognising this fact it is not difficult to conceive that the clinical features which result from this disease may vary enormously. The ordinary types of poliomyelitis as it affects the spinal cord, giving rise to a flaccid palsy of legs, arms, and trunk, are so well known that I do not propose to deal with these, except in so far as they are seen in association with other symptoms, and aid the diagnosis, which otherwise might be obscure.

I. ATHETOSIS OF LEFT ARM AND LEFT SIDE OF FACE, FLACCID PARALYSIS OF LEFT LEG, AND SOME FLACCID PARALYSIS OF RIGHT LEG.

A. A—, æt. 7 years, was the seventh of eight children. She was a healthy baby till the age of ten months, when she had an acute illness with convulsions. From this she recovered, but it left her with paralysis of the left side. She subsequently learnt to talk, was intelligent, and went to a school for physically deficient children. She never learnt to walk.

When first seen she was a pale child, with marked athetosis of the left arm and involuntary movements of the left side of the face. She was unable to

* From the 'British Journal of Children's Diseases.' Revised and enlarged edition prepared for the CLINICAL JOURNAL by the author.

stand, and if an attempt were made to place her on her legs she supported her weight on her right leg with the knee flexed, the left leg being too weak to support any weight. The left hand went into violent spontaneous movements of an athetoid character (Fig. 1) on attempting to walk or make other movements. The child had little or no power of movement over the left hand; there was, however, no rigidity, and the hand, though at times strongly contracted, yet when moved passively was quite flaccid. The left leg showed the typical flaccid palsy of poliomyelitis with wasting of muscles, loss of deep reflexes, and loss of electrical reaction of all muscles below the knee, and to a lesser degree above the knee.

This case illustrates the association of an athetosis with a poliomyelitis. There is no reason to doubt that the paralysis of the legs is due to a spinal lesion and the athetosis of arm and face is due to a cerebral lesion. It may be questioned whether there is sufficient evidence to prove that the condition is due to the virus of poliomyelitis, but so far as the legs are concerned they may be considered typical of the condition. The arm paralysis occurred at the same time, and there seems no reason for hypothesising another toxic condition for the production of the focal lesion in the brain. The contraction which was present in the leg has been corrected, the left leg supported by a celluloid splint to the hip, and the right leg by a celluloid splint to the knee. The girl walked fairly well with some support, but the athetosis of the left arm tended to upset her balance (Fig. 2).

In November 1913, she was transferred to the care of Dr. Pugh, at Queen Mary's Hospital, Carshalton. She steadily improved, and in May, 1915, her condition was such that she could walk quite well alone, with a simple splint on the left leg

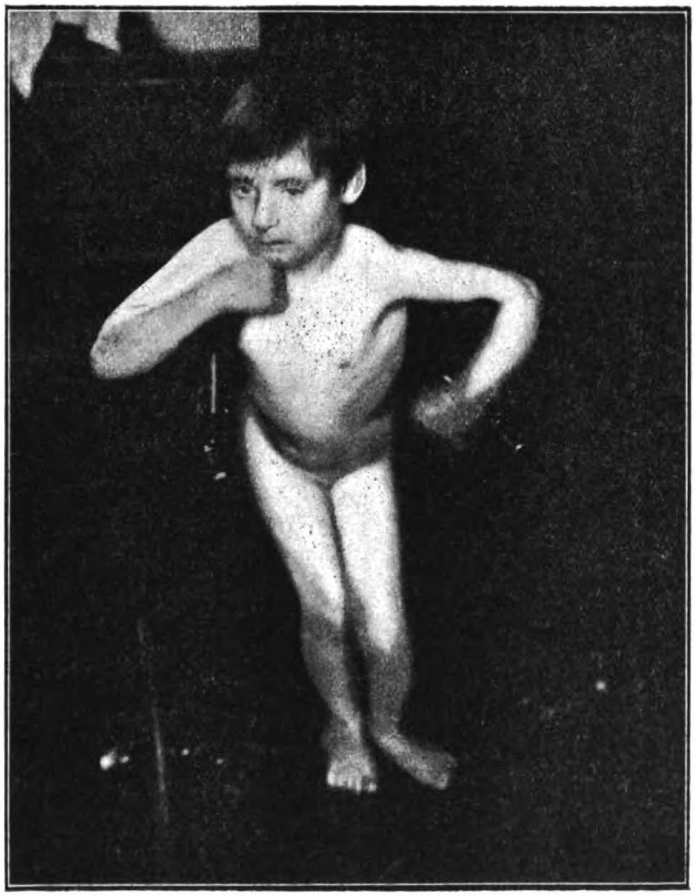


FIG. 1.—Poliomyelitis of legs and athetosis of left hand and face. The left hand is blurred owing to its constant movement.

FIG. 2.—Instantaneous photo. of same child in celluloid splints.

—she could walk without a splint, but owing to the flaccid condition of the muscles of the left leg below the knee, the leg tended to give way.

The athetosis of the left hand remained the same—and in association with this there was marked over action of the left side of the face. The condition of the child in May, 1915, is shown in Fig. 3.

II. POLIOMYELITIS WITH RIGID EXTENSION OF THE LEGS AT THE HIPS.

H. G—, the fifth child of a family of five, was taken acutely ill with poliomyelitis in September, 1912, the limbs, trunk, face and eyes being affected at the onset. He was acutely ill for fourteen days, then gradually recovered the power in his right arm, but had severe pain and tenderness in trunk and lower limbs. When seen in March, 1913, both legs were rigid at the hips and everted, and attempt at movement caused acute pain. Below the knee the muscles were flaccid and the feet were in a position of equinus. The muscles around the left shoulder were completely paralysed, and there was likewise considerable intercostal and abdominal paralysis. The child was quite rigid in the extended position, and if lifted off the bed by the hands placed under the head and heels, he maintained this extended position (Fig. 4). This rigid extension of the hips appeared to be due to the unbalanced action of the gluteal muscles, the psoas and iliacus muscles being completely paralysed. Any attempt at flexion of the hip gave rise to pain, but if once flexed, the hip-joint could be moved in any direction quite easily. X-ray examination made it certain that there was no hip disease. All the deep reflexes were abolished in the lower limbs.

The clinical features of this case suggested the

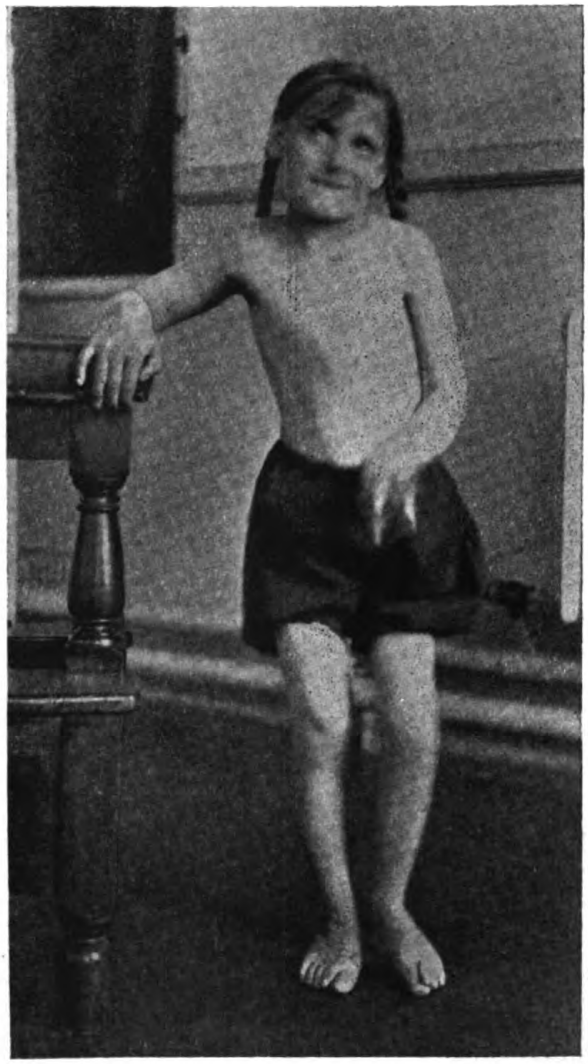


FIG. 3.—Photo. of same child some two years later (May, 1915,) showing the improved condition of the legs. The athetosis of the left hand shows no alteration.

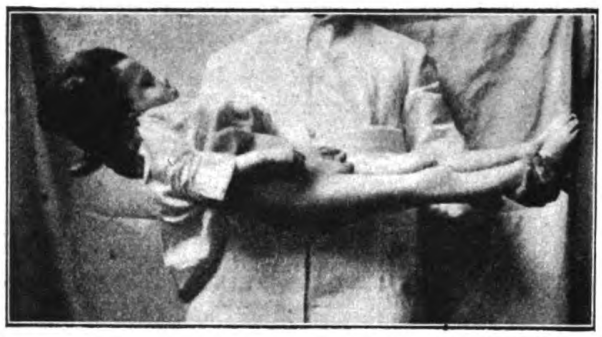


Fig. 4.—Poliomyelitis with rigid extension of legs at hips.

possibility of some disease in the hip-joints, but there seems to be no doubt that the condition was due to an extensive and rather unusual grouping of paralysed muscles due to poliomyelitis. The pain in this case has persisted for over fourteen months. It is much less acute than eight months ago, but movements are still resented and obviously painful.

III. Poliomyelitis with Flaccid Palsy of the Right Arm and Left Leg, and Rigidity of the Right Leg in the Flexed Position.

A. T.—, the second of two children, was quite healthy at birth, but at the age of five weeks had "brain fever" with paralysis of the right arm and left leg. The right leg subsequently became paralysed and in a flexed position at the hip. When seen at the age of four months the child was fairly nourished; had complete flaccid palsy of the right arm and left leg. He lay on his back with the right leg cocked up in the air, the right foot being rotated inwards in a position of varus. The right knee-jerk could be obtained, the left was absent.

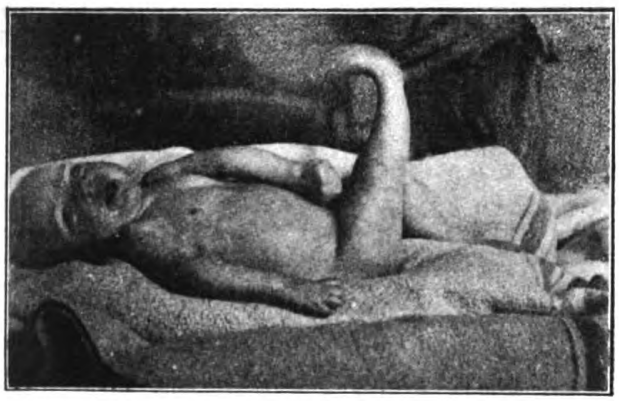


Fig. 5.—Poliomyelitis with rigid flexion of right hip.

On electrical examination no contraction could be obtained in the muscles of the right arm and the left leg, whilst in the right leg the muscles above the knee reacted to faradism, whilst those below failed to react. The condition of the right arm and the left leg was typical of poliomyelitis, and the cocked-up position in the right leg seemed to be due to the contraction of the psoas-iliacus group of muscles, whilst the extensors of the thigh on the trunk, *i. e.* glutei, etc., were paralysed. The flexed condition of the thigh in this case is the reverse of the extended condition seen in Case II.

IV. Poliomyelitis affecting the Muscles of the Neck.

William F—, æt. 11 years, was taken acutely ill about the middle of March, 1915. He had severe headache, vomiting and constitutional symptoms. One week after the onset a "measle-like" rash appeared, which lasted two days, and it was noted that his neck and arm were weak. The case was regarded as one of cerebro-spinal meningitis and notified as such. He was seen by the referee, who recognised the true nature of the case, and sent him to hospital. On admission he was an intelligent, thin, and rather lacrymose boy, quite unable to hold the head up, which lay on the pillow in whatever position it was placed (Fig. 6). He had some weakness of the shoulder muscles, but could perform all movements, the greatest weakness being in the deltoids.

He could move his legs in all directions and could walk so long as his head was held for him, but the sudden falling of the head tended to throw him off his balance. The thoracic and abdominal muscles acted well, and no weakness of the diaphragm could be detected. The deep and superficial reflexes of the lower limbs were equal and active.

There was no affection of any of the cranial nerves, and the mother stated that even at the onset there had been no difficulty in swallowing or articulation. Lumbar puncture was performed, and the cerebro-spinal fluid examined. It was clear, contained ·15 per cent. albumen, no cells, and gave a negative Wassermann.

A plaster-of-Paris splint was made for the neck

FIG. 6.—Photo. showing flaccid paralysis of the neck muscles—the boy could not raise his head and it tended to fall back into the position shown.

so as to keep the head in the erect position. The boy could walk quite well so long as the head was in the splint (Fig. 7).

He gradually improved, gained power in the neck muscles, and by the middle of May, 1915, had recovered considerably ; he still had to be careful about balancing his head, for a slight push would throw it back and it could not recover quickly. With regard to diagnosis there can be little doubt. The acute onset with headache, fever, vomiting, and a measles-like rash certainly suggested the possibility of cerebro-spinal meningitis. The character of the cerebro-spinal fluid negatived that supposition and is compatible with that of poliomyelitis. In extensive cases of poliomyelitis it is not uncommon to find the neck muscles markedly affected, and in such cases bulbar symptoms are often present. The unusual feature of this case is that the neck muscles are extraordinarily involved, whilst those of the limbs, trunk, and bulb have escaped.

Considering the site of the lesion it is interesting to note that the diaphragm shows no paralysis.

V. POLIOMYELITIS OF THE RIGHT ARM AND LEFT LEG OCCURRING DURING INTRA-UTERINE LIFE.

A. S—, the second of three children, was born in September, 1911. The mother was not attended by a doctor. She stated that she did not notice the weakness of the right arm until the child was two months old, but this child, unlike her other children, never made any attempt to move its arm. Wasting of the left leg was noticed some months later, but no attention was paid to this until the child was eighteen months old, when it was found that it could not walk. The child had no acute illness from the time of birth till two months old, when the paralysis was first noticed. The birth was easy, and there was no evidence of injury during parturition. The child was intelligent and well nourished, had a complete flaccid palsy of the right

FIG. 7.—Same boy in a plaster-of-Paris splint supporting the chin and occiput. With this he could walk about in comparative safety and comfort.

arm, with considerable wasting, and a flaccid palsy of the left leg, also accompanied by wasting. The right knee-jerk was active, the left absent. The child presented the typical features of poliomyelitis. The question was whether this condition occurred during intra-uterine life, at birth, or subsequently. It seemed impossible, considering the severity and wide extent of the lesion, that the child could have had an acute attack of poliomyelitis, which should have escaped the mother's attention, between birth and the second month of life. The paralysis of the right arm and leg was not such as one would expect from a birth injury. The case was clinically very similar to that recorded by myself in 'Brain,' 1910 (xxxiii, p. 149), in which a necropsy was eventually performed, and showed changes in the spinal cord characteristic of old poliomyelitis.

VI. Poliomyelitis and Ataxia; probably due to Involvement of the Cerebellum.

R. S—, æt. 5½ years, and M. S—, æt. 1½ years, brother and sister, were both taken acutely ill within a week of one another in August, 1909. The elder child had left facial paralysis and ataxia of his right arm and leg, whilst his sister had paralysis of the left side of the face and flaccid palsy of the neck muscles. In the elder child the ataxia cleared up in about fourteen days after the onset of the acute illness. The paralysis of the face cleared up somewhat later.

In the case of his younger sister the weakness of the face persisted for many months, as did also the weakness of the muscles of the neck, some permanent paresis of the neck muscles remaining. In these two cases there is no doubt that the paralysis was due to acute poliomyelitis, and in the case of the elder child it seems probable that there was a focus of the disease in the cerebellum.

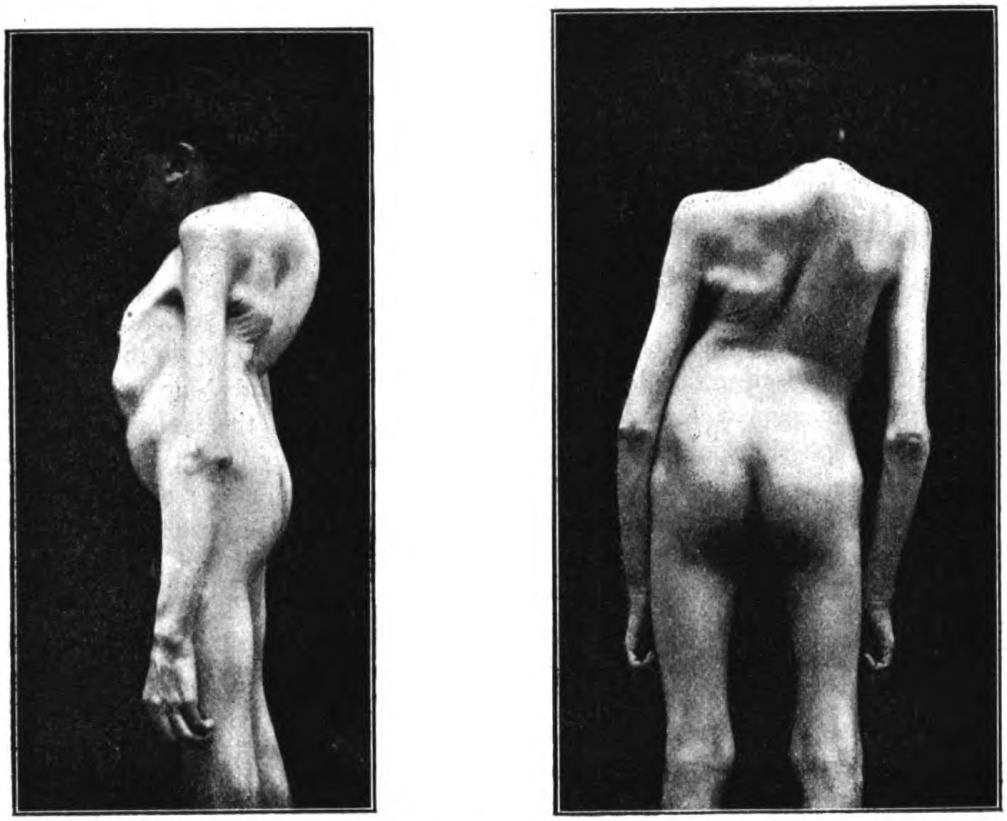
VII. Poliomyelitis and Toxic Neuritis.

It is often questioned whether the virus of poliomyelitis can give rise to symptoms of a toxic polyneuritis. If the presence and persistence of pain are taken as evidence of the neuritic type of the disease, then it may be asserted that there is no justification for the title, since both these symptoms are well recognised accompaniments of affection of the spinal cord. If, however, there is evidence of weakness and wasting of the peripheral muscles which affects all the extremities more or less symmetrically, and at the same time there is definite alteration of sensation in the peripheral portion of the limbs, with tenderness of the muscles, then it will be justifiable to regard such a case as one of neuritis. If such a case occurred with a sudden onset, or in association with typical cases of poliomyelitis in other members of the same family or household, it would be justifiable to assume that the same poison was the causative factor. The recent work of Kling, Petterson and Wernstedt furnishes pathological proof that the virus of poliomyelitis may cause a degeneration of the lower motor neuron not dependent on an infiltration of the tissues. As evidence that poliomyelitis may produce toxic polyneuritis, the following cases may be quoted:

J. F—, æt. 16 years, a milkboy. In July, 1910, he had some pain in both legs. On July 30th he was taken acutely ill, vomited for three days, had pain in the abdomen and legs. On August 3rd, he could not stand, the legs gradually got weaker, and after a few weeks he was absolutely unable to move them, nor could he feel pin-pricks below the knees.

He was in bed for two-and-a-half months, and after the seventh week his legs began to get better; his arms were never severely affected. He came under my care in November, 1910; he had then weakness of the arms, a feeble grasp, and generalised wasting. The trunk muscles were unaffected. The legs were wasted below the knee, he had double foot-drop, absent knee-jerks, and absent ankle-jerks. Electrical changes of the character of R.D. were present in the muscles of the arms and legs. No loss of sensation could be detected when the patient was seen in November, 1910. There was no evidence of poisoning by lead or arsenic. He recovered under treatment, and was discharged well in January, 1911. The case was obviously one of toxic neuritis. The only question which arises is whether his condition could be attributed to the virus of poliomyelitis.

The second case is that of E. H—, æt. 5 years in 1898. He was taken acutely ill at the end of August, 1898; he was feverish, lay prostrate, and was unable to turn in bed. He had acute pain in the back and limbs; he had no affection of the bladder or rectum. He was admitted to the Children's Hospital in October, 1898; he was then



FIGS. 8 and 9.—Poliomyelitis, neuritic type; late effect.

quite unable to walk or sit up in bed. He had considerable pain and tenderness of the trunk and and limbs. He had no cranial nerve paralysis, but his voice was nasal and there was some weakness of the palate. There was a *general* weakness of the legs, arms and trunk; the knee-jerks were absent, there was no loss of sensation, and the muscles on electrical examination showed the changes characteristic of R.D. He gradually recovered and went out of the hospital able to walk, but still very weak. Two years later he was still very weak and had considerable difficulty in rising from the ground and in going upstairs.

He was seen again in 1913. He is now twenty years old; he is very small in stature, 4 ft. 2 in.; he has a marked scoliosis to the right (Figs. 8 and 9). Both arms and legs are weak and thin, but the loss of power is general and not local, and does not look like a residual poliomyelitis. He still has difficulty in rising from the ground and in going upstairs.

The case, when seen in 1898, was regarded as one of poliomyelitis or peripheral neuritis following influenza, which the boy was said to have had; but it is suggested that the virus of poliomyelitis may have been the cause of toxic neuritis, from which he has never made a complete recovery.

CONCLUSIONS.

The above cases form a series illustrating some of the more unusual manifestations of poliomyelitis and serve to indicate some of the various symptoms which may result from an infection by that poison.

September 20th, 1915.

CLINICAL JOTTINGS.

(*Continued from p.* 304.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

On the use of Uranium Nitrate in Diabetes.

Before the value of any drug in the treatment of diabetes can be estimated, it is necessary by means of diet and general management to bring the patient into a condition of physiological equilibrium so far as the excretion of sugar is concerned. When this is arrived at, and no further improvement takes place, the drug can be given. If, then, further improvement follow its administration, and retrogression occurs when it is suspended, to be followed again by improvement when it is renewed, it is fair to conclude that the drug given has had a beneficial effect. This has been repeatedly my experience with uranium nitrate, and therefore I am convinced that it is a very useful drug in diabetes. It is not a specific, nor, indeed, are the other stock drugs, *e. g.* codeia or opium; but it suits some cases in which these other drugs have failed. To produce its effect it must be given in full doses, beginning with a grain two or three times a day, and increasing the dose up to 10 or 20 gr. three times a day. The limit is reached when it begins to disturb the digestion by exciting dyspepsia or diarrhœa. The limit varies greatly in different cases. In some 2 or 3 gr. are all that can be tolerated, in others 20 gr. produce no disturbance. When the limit is reached the dose should be reduced, or its administration suspended for a few days and then resumed. As the action is continued, it is indeed well in every case to stop the drug for a week or so at a time. In this way its use can be continued for months, and with great benefit.

When the drug is doing good its first effect is to relieve thirst, then to reduce the amount of urine and then the amount of sugar. I have more than once seen the sugar greatly reduced, or indeed caused to disappear, so long as the drug was taken, with a corresponding increase in body-weight. Many patients volunteer the statement that they are different persons, so much better do they feel, while taking the medicine.

Why the drug is not more used is due to the fact that it is not administered in the way I have indicated.

As to its real value in certain cases my experience leaves me in no doubt whatever.

Loss of Weight in Visceral Cancer.

The loss of weight in visceral cancer is sometimes astoundingly rapid. Thus I have seen a man of about 12 stone with cancer of the liver lose half his weight in three months, at the rate, that is, of nearly one pound a day. Such rapid emaciation as this is very rare.

Appearances may be deceptive. Thus I remember a woman with cancer of the spleen who was enormously fat for her height, and weighed 16 stone at the time of her death. Yet she had lost 4 stone in as many months.

Loss of weight more or less rapid is, of course, characteristic of cancer, yet patients who are ultimately proved to have visceral cancer by post-mortem examination may retain their weight unaltered for months together, or even after having lost weight may, when carefully nursed, regain much that has been lost. This latter is most frequently seen in cancer of the stomach, where from want of proper assimilation of food, the emaciation of starvation is added to that incidental to the disease. Careful feeding and treatment may remove the former, and so the nutrition is improved and weight increased.

(*To be continued.*)

Radium versus Surgery in Vesical Neoplasms.—Cures in bladder tumours by the use of radium, Squier says, may be hoped for only in benign papillomas; although it may be possible in certain instances to render the symptoms of vesical malignancy less distressing by intra-urethral, suprapubic, rectal or cross-fire intra-urethral and rectal radiations. By the time vesical carcinoma has become clinically manifest the growth has already extended too far to be readily influenced by the amounts of radium at present at command. The treatment of vesical carcinoma is no different from that of carcinoma elsewhere. If the growth is accessible, wide surgical extirpation followed by every means known to treat any unremoved growth or recurrence.—*Journ. A.M.A.*, vol. lxv, p. 743.

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We regret that owing to the war the number of pages must be diminished for a time.

THE INDICATIONS FOR OPERATION IN CHOLELITHIASIS.

By A. RENDLE SHORT, M.D., B.S., B.Sc., F.R.C.S.,

Examiner and Ex-Hunterian Professor, Royal College of Surgeons; Lecturer, University of Bristol; Assistant Surgeon, Bristol Royal Infirmary; Capt., R.A.M.C.

THERE appears to be an unusual degree of difference of opinion in the profession to-day concerning the treatment of gall-stones. Some practitioners look upon a patient with that fateful deposit of cholesterin in his gall-bladder as they would upon one with appendicitis, and hurry him off to a surgeon forthwith; the majority go to the other extreme and with more or less faith prescribe olive oil and salts made in Germany, unless or until dire necessity compels operative interference. The results of some very important investigations have recently been made known which should go far to place our decisions in individual cases upon a sound basis. It is so very tempting and so very misleading to work upon a too narrow experience: "I saw Mrs. A—, for gall-stones; she took olive oil, and now for nine months I have not been called to her again," or "Mrs. B— had an operation and has been perfectly well ever since, and, therefore, I can assure Mrs. C— that she has only to have her stones removed and she will live happy ever after." As a matter of fact it is only a careful study of scores or hundreds of cases well followed through for years that furnishes us with a reliable basis for judgment, and such studies are now available for our use.

First let us clear the ground by tabulating the conditions in which all would agree that operation for gall-stones is urgently indicated as the only means of saving life or rendering it tolerable.

(1) *Many, frequent, severe attacks of gall-stone colic.*—If these are repeated every few months for years, each attack is of grave severity, and the

aforesaid salts and oils work no improvement, of course there must be an operation.

(2) *Persistent jaundice due to stone impacted in the common duct.*—If the jaundice lasts for two or three weeks without abating, and the stools are clay-coloured, removal of the stone is obviously necessary.

The real difficulty in this group is to decide whether the persistent jaundice is due to gall-stones at all. The usual alternatives are simple catarrhal jaundice, certain cases of jaundice associated with splenomegaly, cirrhotic pancreatitis, and especially malignant disease of the pancreas or bile-ducts. Of these, the splenomegalic cases are easily recognised by feeling the spleen, which is enormous. One patient, a woman, æt. 40 years, sent me by Dr. Lauder Smith, of Wells, put me in a difficulty by showing signs of impacted gall-stones and splenomegaly at the same time, but her severe attacks of colic led me to explore, and she was eventually cured after removal of the stones, though the splenomegaly persisted. The differential diagnosis between gall-stones in the common duct and cirrhosis of the head of the pancreas is of no great practical importance seeing that both conditions call for operation. Catarrhal jaundice often occurs in quite young people, there is but little pain, and it passes off in two or three weeks. I have been struck by the frequency with which catarrhal jaundice in young adults leads to the development of biliary calculi in later years, presumably on account of the infection of the gall-bladder.

The following table gives the differential diagnosis between gall-stone in the common duct and cancer in patients with jaundice persisting several weeks.

Gall-stone in Common Duct.	Cancer of Pancreas or Bile-Passages.
(1) Severe attacks of pain.	No.
(2) Tenderness of gall-bladder.	No.
(3) Gall-bladder not enlarged.	Palpably enlarged.
(4) Obstruction not complete, therefore jaundice varies, and a little bile occurs on some days in the stools.	Jaundice increases to a deep so-called "black jaundice."
(5) No wasting.	Marked wasting.
(6) —	Stools contain excess of fat and meat fibre.
(7) —	Finally, nodules in liver; ascites.

Of these, the signs to trust are the history of attacks of severe colic with tenderness on the one hand, and the "black jaundice" on the other, especially if wasting is coming on. I was asked by Dr. Colston Wintle to see a man with a fortnight's jaundice who combined all these signs; he proved to have a gall-stone as well as cancer at the operation. In many cases where there is doubt, the rapid advance of symptoms in cancer will clear up the diagnosis after waiting for a week or two.

If there is a reasonable prospect of finding a gall-stone, operate. If all the evidence points to malignant disease, it might be reasonable to explore "on the off chance," but if cancer is found the patient will probably die in a few days or weeks and the surgeon is likely to get the blame for killing him, unless the friends are honest and intelligent people and thoroughly understand the situation.

(3) *Hydrops of the gall-bladder.*—In this case there is a stone in the cystic duct; the gall-bladder forms a large palpable tumour, and there are or have been attacks of pain. The gall-bladder needs removal, of course, with the stone.

(4) *Empyema of the gall-bladder.*—Here a hydrops has become infected and causes fever and tenderness as well as a tumour. It should be drained at once.

(5) *Perforation of the gall-bladder by a stone causing peritonitis.*

(6) *Intestinal obstruction by a gall-stone.*

(7) *Acute cholecystitis.*—This dangerous condition may present itself under two slightly different forms. Starting as an ordinary attack of gall-stone colic, it may, after a few hours or next day, give rise to fever and tenderness in the right hypochondrium lasting for several days, or, on the other hand, the pain may be less severe and not colicky, but the tenderness and fever be present from the first. In fact, the condition closely resembles an acute attack of appendicitis, except that the tenderness is in the hypochondrium instead of in the iliac fossa. Only this week I was summoned to see such a case diagnosed by the doctor in attendance as appendicitis—a very common mistake. A certain number of these patients die of peritonitis following perforation of the wall of the gall-bladder.

If operation is performed, one usually evacuates a thin muco-pus mixed with the stones; the last gush is often pure pus. When, as often happens,

there is a palpable tumour, the case merges into one of empyema of the gall-bladder.

Now, with regard to surgical treatment, in certain types of this condition it is quite clear that one ought to operate, and in certain other types it will be better to wait for a more favourable occasion. Without doubt to interfere in the presence of septic complications is a hazardous business. Two short series of such cases reported by the writer from the records of the Bristol Royal Infirmary, and by Mr. D'Arcy Power from his own practice at St. Bartholomew's Hospital, show a death-rate of nearly 50 per cent., and in 520 cases operated on by Kehr the mortality was 45 per cent. These figures include a small proportion of cases with perforation of the gall-bladder and peritonitis. Apart from this a death-rate of 24 per cent. is given by the Mayos in 102 cases. We may take it, therefore, that if it is necessary to operate for gall-stones in the presence of sepsis, about one in three will die of liver abscess, peritonitis, chest complications, etc. That being so, one should only interfere when the patient's life is in present danger, that is, when the fever and tenderness

(*a*) Come on apart from a definite attack of gall-stone colic, or

(*b*) Are associated with colic, but persist three or four days without improvement, or

(*c*) There is a palpable empyema of the gall-bladder as large as an orange (which shows that drainage through the bile-ducts is blocked), or

(*d*) If signs of peritonitis develop.

On the other hand, what appears to be a typical attack of gall-stone colic complicated by fever should not be operated on at the time, in the absence of the four danger signals just mentioned. It will probably get well and the dangers of the operation will be much less after a few weeks of quietness.

(8) In the first six conditions passed under review, the indications for surgery were clear. The seventh condition, cholecystitis, called for more consideration. We must now inquire about another disputed problem: ought one to operate after a few attacks of hepatic colic occurring at fairly long intervals? Ought a gall-stone, in fact, to be treated like an appendix liable to periodical outbursts of inflammation?

We remove the appendix during a quiet interval after one, two, or more attacks, for two reasons: first, that at least half the patients will have further trouble, which will lay them up often at very inconvenient times; and secondly, because any attack may lead to perforation and death.

The early removal of gall-stones is to be advocated, because these arguments are just as cogent, and also for a third reason, that early operation, as practised in America and Germany, is safer and gives far better end-results than the late operation which is the prevailing fashion in England. These three arguments we must now proceed to defend.

First, a patient who has once or twice suffered from gall-stones will probably suffer again. The victim fondly hopes the stone has "passed." Sometimes he proudly produces it from the next evacuation of the bowels, and does not realise the ominous significance of those facets where it rubbed against its brothers. Usually no stone can be recovered from the motions. The mere presence of the calculus proves the existence of chronic cholecystitis, and the fact remains that efficient curative drainage of the gall-bladder cannot be obtained down the narrow cystic duct. Admittedly the acute symptoms may only come on at long intervals and occasionally they seem to cease for ever, but surely all experience goes to show that the following is the common history: A lady, well known to the writer, who twenty years ago had a typical first attack, then recurrent spells of vomiting and pain, which passed undiagnosed, occurring at long intervals, now during the past four or five years desperately severe illnesses with fever and jaundice every six or nine months. Many patients reach this last stage much sooner.

Secondly, the occurrence of gall-stone colic is a menace to life. True, the patient seldom dies in a first attack. The dangers are as follows: In about 30 per cent. of the cases which are sufficiently severe to resort to a hospital or a consulting surgeon, a stone becomes impacted in the common bile duct or cystic duct, and produces in the former instance persistent jaundice, and in the latter an enormously dilated gall-bladder. This is proved by the figures of the Mayos, Kehr, and the Bristol Royal Infirmary. It is not suggested that 30 per cent. of *all* sufferers from gall-stones come to such a pass, but 30 per cent. of those who have severe or frequent attacks. These conditions, of course, necessitate an operation, because septic complications will most probably prove fatal sooner or later if the obstruction is not relieved.

Next, there is a genuine danger, not sufficiently realised, of the development of cancer of the bile passages. Various large statistics show that this closes in the scene in about 8 per cent. of the sufferers from gall-stones.

Again, the risks of an attack of acute cholecystitis are by no means negligible. Certain rare complications of gall-stones, such as acute intestinal obstruction from impaction of the stone in the bowel, perforation of the gall-bladder, subphrenic abscess or suppurative pylephlebitis, taken all together, raise the death-rate, it may be by 2 or 3 per cent. The *early* risks are less than in appendicitis; the *late* risks are greater.

The current English practice, after one or two definite attacks of gall-stone colic, has been to treat with olive oil, if the patient is willing and able to take it, or by dieting and mineral waters, until the frequency of the periods of agony or invalidism, or the onset of one of the above-mentioned urgent indications, makes the patient as well as the doctor desperate, and willing even to brave the terrors of an operation rather than face another attack.

Olive oil is very, very uncertain; occasionally one hears of cures, but it is difficult to understand why it should do good. The theory which led to its use was that since a gall-stone dissolves in olive oil, some of it might get up into the gall-bladder and perform useful service, but food-stuffs do not invade the bile passages.

The point to which we must now direct attention is that this waiting until matters are desperate increases the risk of the operation, and lessens the chances of permanent relief therefrom. In America and Germany, where the practitioners and surgeons are much more ready to recommend early removal of the stones, the death-rate in ordinary cases, without septic or other complications, is about 2·5 to 3 per cent.

In England, the death-rate appears to be about 10 to 11 per cent. (D'Arcy Power, Bristol Royal Infirmary) in hospital practice. The reason is not so much superior skill across the water, but earlier operation. Thus of the Mayos' cases, 102 out of 1500, or 6·8 per cent., showed grave complications; of the English cases, 32 out of 157, that is 20 per cent. No doubt English private practice might show better results, but figures do not appear to be available. D'Arcy Power points out that in a series of 27 uncomplicated cases he had no deaths; on the other hand, of 19 Bristol patients with extensive adhesions, 30 per cent. died. Adhesions, of course, mean longstanding mischief of a severe type.

Not only does waiting until matters become desperate increase the risks of the operation, but it also lessens the prospects of permanent cure. The English cases (D'Arcy Power, Bristol Royal Infirmary) show 15 to 20 per cent. unrelieved; the figures of Stanton from Ochsner's American clinic, and of Arnsperger of Heidelberg, show only 6 to 7 per cent. unrelieved.

TABLE I.—*The Operation Mortality for Gall-stones.**

Author.	All cases.		Stones in gall-bladder.		Duct cases.		Serious complications.	
	No.	Died.	No.	Died	No.	Died	No.	Died.
		%		%		%		%
D'Arcy Power	73	23·3	53	11·3	—	—	20	55
Kehr . .	1600	16·5	691	2·9	389	3·3	520	44·6
Mayo . .	1500	4·4	1164	2·5	105	2·9	102	24
Bristol Royal Infirmary .	84	13	31	9·6	27	11	12	42

* The tables are from the article on gall-stones by the author in 'Index of Prognosis and End-results of Treatment.'

TABLE II.—*Prospects of Relief after Operation for Gall-stones.*

Author.	Number traced.	Cured.	Better.	Unrelieved.	Incisional hernia.	Second operation.
		%	%	%	%	%
Arnsperger . .	147	64·6	19·7	6·9	8·8	?
Stanton . .	245	79	15	6	3	2
D'Arcy Power .	20	70	—	20	10	0
Bristol Royal Infirmary . . .	59	52·5	32·2	15·2	5	6·7

What are the causes of this lack of success? Principally three: adhesions, recurrence of stones, or incisional hernia. Adhesions may give rise to symptoms exactly like attacks of hepatic colic, and second operations show that this is a commoner cause of recurrent trouble than re-formation of the calculi. Of 19 Bristol patients with extensive adhesions at operation, 36 per cent. were unrelieved, whereas in cases without adhesions

only 10 per cent. complained of further trouble (mostly incisional hernia).

We learn from all this that operation for gall-stones after a few attacks, before septic complications have supervened or adhesions formed, has a mortality of about 3 per cent., and only 7 per cent. are unrelieved, but that because the English custom is to wait for more trouble, the actual mortality in hospital practice is 10 per cent. in ordinary and nearly 50 per cent. in septic or perforated cases, and 15 to 20 per cent. of the survivors fail to obtain comfort in the years following.

We see, therefore, that the indications for operation after a few attacks of gall-stone colic are nearly, if not quite, as strong as those for interval appendicectomy, and a practitioner who looks to his patient's future interests must advise operation, in most cases, at an earlier date than is now customary. As a fair working rule, I should advise operation after one febrile or two afebrile attacks, except in aged, feeble, or bronchitic subjects.

Summary.

A. Operation is to be urged upon patients with any of the following :

(1) Very frequent or severe attacks of gall-stone colic.

(2) Jaundice due to stone in the common duct, persisting over two to three weeks. .

(3) Palpable swelling of the gall-bladder.

B. Immediate operation is to be urged :

(4) For perforation of the gall-bladder, with oncoming peritonitis.

(5) Intestinal obstruction by a stone.

(6) Acute cholecystitis ; if preceded by an attack of hepatic colic, operate after three or four days ; if characterised by fever, tenderness, guarding and palpable gall-bladder but no colicky pain, operate at once.

C. Operation is desirable, but not exactly urgent :

(7) During a quiescent interval, a few weeks after one severe febrile attack, or two afebrile attacks of gall-stone colic, if the patient is in other respects a fit subject for surgical intervention.

September 27th, 1915.

A CLINICAL LECTURE

ON

DISEASES OF JOINTS.*

By C. BRAINE-HARTNELL, F.R.C.S.Ed.,
Surgeon to the Cheltenham Hospital.

The first case I wish to show you to-day is a young man whom Dr. Collins referred to me three years ago with tuberculosis of the wrist-joint. He also was suffering from pulmonary tuberculosis. The wrist showed all the signs of tubercular arthritis. You will notice on both flexor and extensor surfaces the scars of healed sinuses. There is still limited movement, and the wrist is not sufficiently dorsi-flexed, but the disease is soundly healed. The joint was at first fixed on a Lister's wrist-splint, and afterwards in plaster of Paris. During the treatment Dr. Collins gave repeated doses of tuberculin, but the results were not encouraging. In spite of all treatment the joint made no attempt to improve and became so bad that the question of excision was considered. As a last resort, I applied a Bier's bandage to the upper arm, beginning with two hours a day and increasing the length of the application until 22 hours out of each 24 was reached, the wrist, of course, being fixed on splints. From the commencement of this treatment improvement set in. The bandage was used for six months. The patient, who is a clerk, has been at work for many months and expresses himself as feeling very well. The interest in this case is the improvement that set in after adopting Bier's hyperæmic treatment. Mr. Robert Jones tells me that he has not seen good results from Bier's bandage in tubercular arthritis, though he is a believer in it for septic cases. The next point in this case is that, although the patient has recovered with a useful joint, it is not as good as it should be. It is all-important in injuries and diseases of the wrist-joint to keep the hand in a position of dorsi-flexion, as upon this the strength of the grasp depends. Lister's splint does this to a certain extent, but there is no splint that I know so effectual as Mr. Robert Jones's "cock-up" splint, which I have here, and which I always use now.

* Post-Graduate Lecture delivered at the Hospital.

The next case is a farm labourer, who had a lacerated wound over the dorsum of the third metacarpal bone. The wound was horribly septic, and I expected the extensor tendons would slough. The wound was enlarged and mopped out with carbolic acid 1 in 5 in spirit. A second incision was made over the reddened and œdematous extensor surface of the forearm, and the muscles appeared to be pale and œdematous. The hand was fixed in a cock-up metal splint, kept in a continuous arm bath, and 24 hours after the incision had been made a Bier's bandage was applied. As an operation causes a certain amount of auto-inoculation it is always advisable to allow some time to elapse before applying a Bier's bandage. The patient at once began to improve, the temperature fell, and as you see now the wounds are soundly healed, and he has perfect use of his hand and wrist. If the improvement had not been immediate I should have asked Dr. Hebblethwaite to prepare a vaccine, but this was not necessary.

In this bed we have a man, æt. 47 years, who for the past 8 years has had a "weak knee." You will notice that the joint is very distended with fluid. The movements, instead of being limited, are excessive. There is no pain. If you examine the joint you will find a large loose body or osteophytic outgrowth above and to the inner side of the patella—and there is considerable grating on movement. The knee-jerks vary, sometimes they are absent, at other times present. There is well-marked strabismus, but no Argyll-Robertson pupil, and Mr. Pike reports no optic atrophy. There is no loss of sensation to heat and cold. Here we are evidently dealing with a neuropathic joint, probably a Charcot, though the symptoms of tabes are not well marked. In syringomyelia we also get neuropathic joints, but in this case the joints of the upper limb are generally affected, as it is the cervico-dorsal portion of the cord that is diseased. Professor Howard Marsh has pointed out that in syringomyelia the joints may suppurate, also that scoliosis is often present. Both these symptoms are absent here

Owing to the great bony destruction that takes place in these cases any operative measures are out of court. The only thing we can do is to support the joint in some way. You will see that I have fixed this man with a Thomas's knee-splint, and he gets about on crutches.

In the next bed is a boy, æt. 13 years, who has had disease of his right knee-joint for 3 years. The joint is full of fluid, and is on the point of bursting in one place. It is very painful. Movement is limited, and there is considerable muscular wasting. Here is obviously a case of advanced tubercular disease chiefly affecting the synovial membrane. It is also plain that the joint has not been kept fixed long enough or thoroughly enough to arrest the disease, though he says that at one time he was placed in plaster of Paris. I would suggest here absolute fixation, after aspiration, the use of Bier's bandage and tuberculin. It may be necessary to do an arthrectomy. In a boy of this age an excision should not be performed. It is not advisable to do an excision before the age of 16 or 18. When the disease is arrested the boy should be made to wear a Thomas's knee-splint for at least two years. You may ask, how can we tell when a joint is cured? I know no better answer to this question than the one given by that master in orthopædic surgery, Mr. Robert Jones: "A joint is cured of disease when the range of movement does not diminish by use, or in the case of an ankylosed joint when the angle does not change after use."

Here is another boy with tubercular disease of the knee-joint, but presenting different physical signs. There is pain, limitation of movement, and muscular wasting, but very little effusion. On the other hand, there is considerable enlargement of the lower end of the femur.

There is some backward displacement of the tibia. The three deformities that we are liable to meet with in tubercular disease of the knee are:

(1) Backward displacement.

(2) Outward rotation of tibia.

(3) Genu valgum.

All these must be guarded against.

If you wish to apply extension to a knee-joint, flex the thigh first, and then apply extension. Prof. Howard Marsh has pointed out that in cases like this, with little effusion and considerable bone enlargement, there may be difficulty in eliminating a sarcoma.

The next patient is a man who was shot through the left knee at Dickebuch last November. When he came into the hospital he had an acute arthritis, with a high temperature and rapid pulse, and there was great tenderness over the head of the tibia,

with some œdema. I made an incision on either side of the patella, let out the pus, and washed out the joint with 1 in 40 carbolic. I also turned up a flap over the internal tuberosity of the tibia and drilled the bone, but found no pus there. A drainage tube was left in the joint, passing from one opening to the other, and the joint frequently irrigated. The improvement was immediate. The pain, which had been unbearable, disappeared on the following day. When all the discharge had ceased and the wounds healed he was allowed to get up, but this at once was followed by pain and swelling of the joint and some œdema on outer side of the leg over the head of the fibula. Some thin semi-purulent fluid was let out and the leg again fixed on a back splint. He is now getting about with a stiff knee, but as movement causes no pain or swelling we may consider that the inflammation has ceased. I do not intend to try and break down these adhesions. There is now definite fibrous ankylosis, and I am sure the adhesions are far too massive to be dealt with in this way. If one could break them down they would only form again. There is one other point about this case: at the height of the inflammation there was considerable enlargement of the lower end of the femur, which has now practically disappeared. I hope in course of time he will get more movement in the joint.

Now there are two knee-joints here, both of which show the importance, first, of aspirating joints that are full of fluid, and second, getting a radiogram of all joint injuries in order to make a diagnosis.

The first is in a woman who was admitted under my care 12 months ago, having injured her knee when getting over a stile. On admission there was considerable pain in the knee and great synovial effusion—so much that it was not possible to make a definite diagnosis. The X rays showed a fracture of the internal tuberosity of the tibia with displacement backwards and inwards. The patient refused operation and left the hospital with a stiff knee. When the joint effusion cleared up it was evident that the tubercle of the tibia was not in the mid-line of the joint, and there was considerable enlargement of the internal tuberosity. Although this patient left the hospital with a stiff knee, and I expected her always to have one, you will notice that her movements are now quite normal.

The second patient had also a large effusion into the knee-joint that obscured the diagnosis. As this went down, it appeared that there was a transverse fracture of the patella. Unfortunately, as Dr. Meyrick Jones has joined the R.A.M.C., I could not get a radiogram. I advised an operation and found, to my surprise, not a fractured patella, but that the patellar ligament had been torn away from the patella, and the joint filled with unhealthy looking fluid and blood-clot. I fixed the ligament as well as I could to the patella and surrounded the joint with a bronze wire, passing it through the patella ligament and quadriceps tendon. The outlook is not so good as in cases of fractured patella.

Both these cases, I think, demonstrate the importance of aspirating joints that are acutely distended, in order to assist the diagnosis and relieve this tension on the ligament.

Now let me show you a case of early tuberculosis of the right hip-joint in a girl, æt. 13 years. This joint is more often affected than any other, with the exception of the spine. It usually occurs in the first 10 years of life, and is nearly always a monarticular affection.

The typical symptoms are pain, lameness, wasting of muscles, and limitation of movement.

This girl complained of pains but no lameness, there is slight muscular wasting and definite limitation of movement. It is most important to note that this limitation of movement must be in every direction and not only in one. Disease of the lumbar spine will cause limitation of flexion and extension, but not of abduction and adduction, both of which are well marked here.

With the legs lying parallel when the patient was admitted, the right anterior superior iliac spine was a little lower than the left, thus showing that the limb was abducted, for to bring the anterior superior spines on the same level you must abduct the limb, and conversely, if the anterior superior spine on the diseased side is higher than on the sound side, you know that the limb is adducted. Always test the range of movement by moving the sound limb first. The sound limb in this case can be brought over the diseased one, but the diseased limb cannot be crossed over the sound one to the same extent, therefore adduction is limited.

Next take the question of flexion. In a normal person the lumbar spines and the popliteal space

should lie on the same plane. Here, while the popliteal space is resting on the bed, the lumbar spine is in a condition of well-marked lordosis. To correct this lordosis we must flex the diseased limb. Again, if you flex the sound limb, you will notice the diseased limb is raised off the bed as the lordosis disappears. In order to measure the amount of flexion or lateral displacement, if you will refer to Kingsley's or Lovell's table in any book of orthopædic surgery, you will find tables giving the methods to be adopted in each case. By means of a tape measure and comparing the real shortening, *i. e.* from the anterior superior spine to interior malleolus, with the false shortening, *i. e.* from the umbilicus to the malleolus, you can immediately tell the position of the limb; if the real shortening is greater than the false the limb is adducted and *vice versâ*.

If abscesses form in connection with tubercular joint disease they should be left alone unless causing symptoms which demand interference; then they should be aspirated. Abscesses may occur early or late, and all observers are agreed on the important fact that even large abscesses may disappear spontaneously if the joint affected be rigidly fixed; we have all seen pressure paraplegia in Pott's disease clear up under rest.

As regards treatment I now show you Thomas's abduction frame for these cases. It is a most excellent splint, and nothing else is used, I believe, either at the Liverpool Children's Hospital or at Baschurch. It is easy to apply extension and most easy to wash or move the patient. At present I am treating the patient by extension in the abducted position, as I have not an abduction frame to fit. Some surgeons rely upon weight extension with a flexed hip and the limb extended in abduction or adduction, as the case may be, and gradually brought down to the straight position. I do not think it matters much which method you adopt provided that you fix the affected hip and keep it at absolute rest. Rest, fresh air, and tuberculin should be the main points in treatment. In advanced cases with sinus formation, adduction of the limb and deep abscesses, Mr. Styles is in favour of removing the head of the femur. On the other hand, Sir A. Bowlby reports in the 'Brit. Med. Journ.' for 1908 that in 900 cases he has never done an excision of the hip. As a general rule, avoid operating if possible. Excision and amputation for tubercular disease are a confession of failure.

If the disease is diagnosed early and thoroughly treated, extreme measures will rarely be necessary in the young. In elderly people the case is different.

To rectify deformities osteotomy, either trans- or sub-trochanteric, may be necessary and also division of tendons.

Dr. Gauvain, of Alton, makes great use of plaster of Paris in all tubercular cases and follows this up when the disease is arrested with celluloid supports, and in his hands the results are excellent. Personally I prefer the various splints I have shown you to-day, all of which were made by Messrs. Critchley, of Liverpool.

There is a young woman under the care of my friend, Mr. Howell, who was under me two years ago for pain in the spine, and I was not then able to eliminate early tubercular disease. To-day I can safely say, and Mr. Howell agrees with me, that there is no organic disease present. You will notice there is no kyphosis or scoliosis, and no muscular wasting. The girl can flex and extend her spine, and if the pelvis be fixed she rotates her spine well in both directions. These points eliminate Pott's disease, and we may put the disease down as "functional" and encourage her to get up and about.

September 27th, 1915.

Epigastric Route for Pericardiac Paracentesis.—G. Blechmann decides in favour of the sub-xiphisternal route as advocated by Marfan in 1911. The extreme tip of the xiphoid cartilage is found by the index finger of the left hand, and the trocar is introduced under this, care be taken to keep exactly in the middle line. After passing along the posterior surface of the xiphisternum for a distance of 2 cm., the point of the needle is directed obliquely backwards and upwards until the pericardium is reached. It is claimed that this method reaches with the least danger and difficulty that part of the pericardial sac in which most fluid accumulates. In one patient the author successfully tapped the pericardium 17 times.—*Brit. Journ. of Child. Dis.*, p. 183.

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We regret that owing to the war the number of pages must be diminished for a time.

THE USE OF SMALL DEEP SKIN GRAFTS.*

By JOHN STAIGE DAVIS, M.D.,

Instructor in Surgery, Johns Hopkins University, Baltimore.

THE rapid healing of large granulating wounds is a matter of great economic importance, especially at this time. I wish to call attention to the value of very small grafts, especially to those which contain more of the true skin than the superficial grafts advocated by Reverdin. I have called these grafts "small deep grafts."

The hastening of the healing of granulating wounds by the use of small detached bits of skin was first demonstrated by J.-L. Reverdin.(1) His report to the Société Impériale de Chirurgie, Dec. 8th, 1869, caused universal interest.

He showed a patient on whom he had practised "epidermic grafting," and described the grafts as consisting of the epidermis only. He says, "I raised with the point of a lancet two little flaps of epidermis from the right arm, taking care not to cut the dermis."

Grafts obtained by Reverdin's method are usually thought of as pure epidermic grafts, and he always referred to them as *greffe épidermique;* but in his exhaustive paper (2), published in 1872, he says: "The title 'epidermic grafts' is not perfectly correct, as the transplanted bit is composed of the whole epidermis and a very little of the dermis."

Reverdin also said that if the epidermis alone could be transplanted, the same result would be obtained as when a part of the dermis was included. This, of course, is true as far as the epithelium is concerned, but it has been my experience in a large number of cases that the grafts which are

* By kind permission of the 'Journ. of the American Med. Assoc.' Edition prepared for the CLINICAL JOURNAL by the Author.

somewhat deeper and contain more of the true skin give a more stable healing, and the final result is more like the normal skin than when the thinner grafts are used (Fig. 1).

It is interesting, when we consider that Reverdin developed his method of grafting before the introduction of antisepsis and asepsis, and that his is probably the only type of graft which could have given satisfactory results under such conditions.

The favourable results obtained by other surgeons in England, America, Germany and France, from Reverdin's method, stimulated further investigation along this line. His tiny grafts were gradually superseded by much larger grafts of the same thickness, advocated by Ollier(3) in 1872, and later by Thiersch(4) in 1886. After a few years the value of the Reverdin method seemed to have escaped the attention of most surgeons.

Use of Skin Grafts.

As there is little difference, except in thickness, between the "small deep grafts" and Reverdin grafts, the following methods of preparation, etc., are applicable to either.

Surface on which grafts may be placed.—The grafts may be placed on fresh wounds, but are usually applied to granulating surfaces. The success of small grafts depends to a large extent on the condition of the granulating wound, and it is most important that the granulations be clean, firm, rose pink in colour, and not exuberant. Time is saved in the end by careful attention to this point. It is of advantage to place these grafts on a granulating surface, as there is no pain in the preparation of the surface; no danger of stirring up infection, and no loss of blood, which is an important point in cases already much depleted.

Method of preparing healthy granulations to receive grafts.—On the day preceding the operation all secretions and crusts are removed. The granulations are painted with tincture of iodine and dressed either with balsam of Peru and castor oil, 1 : 3, or with moist boric or salt gauze. The dressings are removed at the time of operation, and the wound washed carefully, without causing bleeding, with sponges dipped in warm salt solution. The surface is dried thoroughly and a pad of dry gauze placed over the wound; it is pressed down firmly

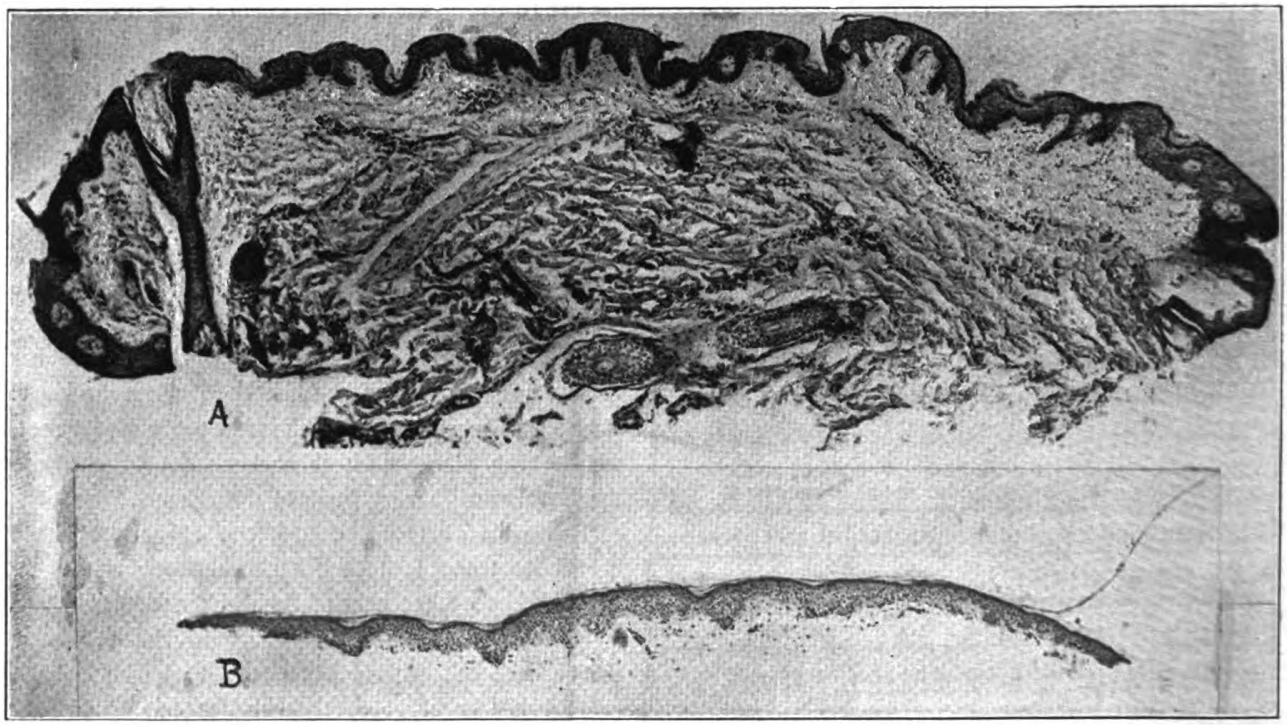

Fig. 1.—Photomicrographs of the same magnification, showing the comparative thickness of a small deep graft and a Reverdin graft. A. A small deep graft cut to include a considerable thickness of the true skin; in the section the edges of the graft are curled inward. B. A Reverdin graft cut to include as little of the dermis as possible. There is a thin layer of the dermis throughout, which is thickest in the centre.

on the granulations and removed only when the operator is ready to apply the grafts. It is most important that the surface to be grafted should be perfectly dry, as the grafts adhere much better to a dry surface, and are less liable to be subsequently displaced.

The source of grafts and area from which they are obtained.— Autografts are almost uniformly successful when placed on a granulating surface which is in proper condition. Isografts are also successful in many instances, but there are wounds which have come under my observation on which no isograft would take, even after repeated trials from many donors. Autografts on the same wounds healed promptly. The grafts may be obtained from almost any portion of the skin. It is considerably easier to cut them from a region such as the front of the thigh (where the skin is quite taut), than where it is lax, as for instance on the abdomen. The region from which the skin is taken has little, if any, effect on the success or failure of the grafts. A large number of these grafts can be obtained from a comparatively small area, thus making it possible to use small grafts on extensive wounds without any severe tax on an ill patient. I have applied several hundred of these grafts at a single operation.

Preparation of area from which grafts are taken.—A satisfactory method of cleaning the skin before removing the grafts is to shave, and then wash thoroughly with green soap and water. Rinse with sterile water and sponge with ether, followed by alcohol; finally rinse with normal salt solution and dry. It is better to use no antiseptic in the preparation of this area, although very satisfactory results follow the use of iodine technique.

Anæsthesia.—As these grafts are obtained with little pain and are usually applied to undisturbed granulations, general anæsthesia is unnecessary, unless the patient is a young child or in a highly nervous condition. It is as a rule desirable to inject 1 per cent. quinine and urea hydrochloride solution, or 0·5 per cent. novocaine, with a small amount of epinephrin, and take the grafts from the anæsthetised area. The infiltration seems to have no hindering effect on the healing of the grafts.

Technique.

The simplest way to obtain these grafts is to pick up a bit of the epidermis with a straight intestinal needle held in an artery clamp (Fig. 2). It is raised so that a little cone is formed, and the base of the cone is cut through by depressing the blade of the knife. The use of forceps and scissors in obtaining small grafts is not desirable, as the skin is thereby injured unnecessarily.

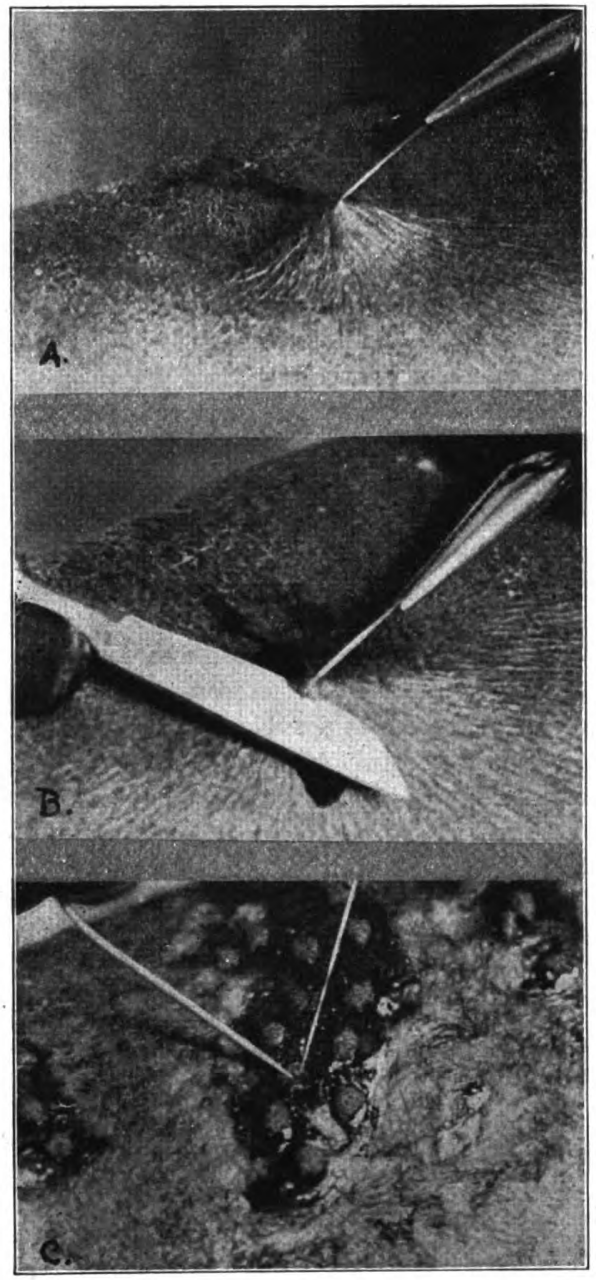

Fig. 2.—A. The straight intestinal needle, held in an artery clamp, with its point engaged in the skin. The skin is raised up in a little cone. B. The scalpel cutting through the base of the cone. C. Placing the graft on a granulating surface. A large opened-out safety-pin is very useful in detaching the grafts from the needle. In this photograph none of the grafts have as yet been pressed down on the wound, and the edges are still curled under.

(The thinner Reverdin grafts may be obtained by cutting off the tip of the cone.) The graft, still on the needle, is transferred to the wound, with raw surface downward. The grafts are placed in rows at intervals of not more than 5 mm. When two rows are in place, a strip of dry sterile rubber protective about 1·5 cm. wide is applied over them so that the lower edge of the protective just covers the lower row of grafts. Then the protective is pressed down over the grafts firmly with a gauze pledget, and the edges of the grafts will uncurl and spread out evenly on the wound. The next row of grafts is placed close to the edge of the protective, and after two or three rows are applied, they are covered with a second strip of protective which overlaps the first piece about one half its width. The protective is pressed down firmly and the procedure is continued in this manner until the whole wound, or the part selected, is covered. The overlapping strips of protective allow the escape of any secretions from the wound into the overlying dressings. The ends of the protective strips which extend beyond the wound edges may be fastened securely to the normal skin by means of a few drops of chloroform. Moist salt gauze over the protective strips secured by a bandage is a satisfactory dressing. The part should also be immobilised as far as possible.

The area from which the grafts are taken may be dressed with boric ointment spread on a sheet of rubber protective, which is snugly strapped over the wound, or with silver-foil and dry gauze.

The grafts are either round or irregularly oval. They vary between 2 and 4 mm. in diameter (Fig. 3), and should be seldom larger than 4 mm. They are thickest in the centre and taper off toward the edges. With a sharp knife the size, contour and depth can be judged accurately.

The wounds left by the removal of the grafts have the appearance of small pits punched in the skin, there being a rim of untouched epithelium between the pits. Fat may be seen in the bottom of some of the pits, which shows that a considerable depth of the skin is used. More bleeding follows the removal of these small deep grafts than when the Reverdin grafts are cut, and the resulting scars are also somewhat more pronounced. Sometimes, when the grafts are small, and the skin blood-stained, it is difficult to determine which is the raw surface. This difficulty is obviated when the grafts are obtained from a negro or when iodine technique is employed.

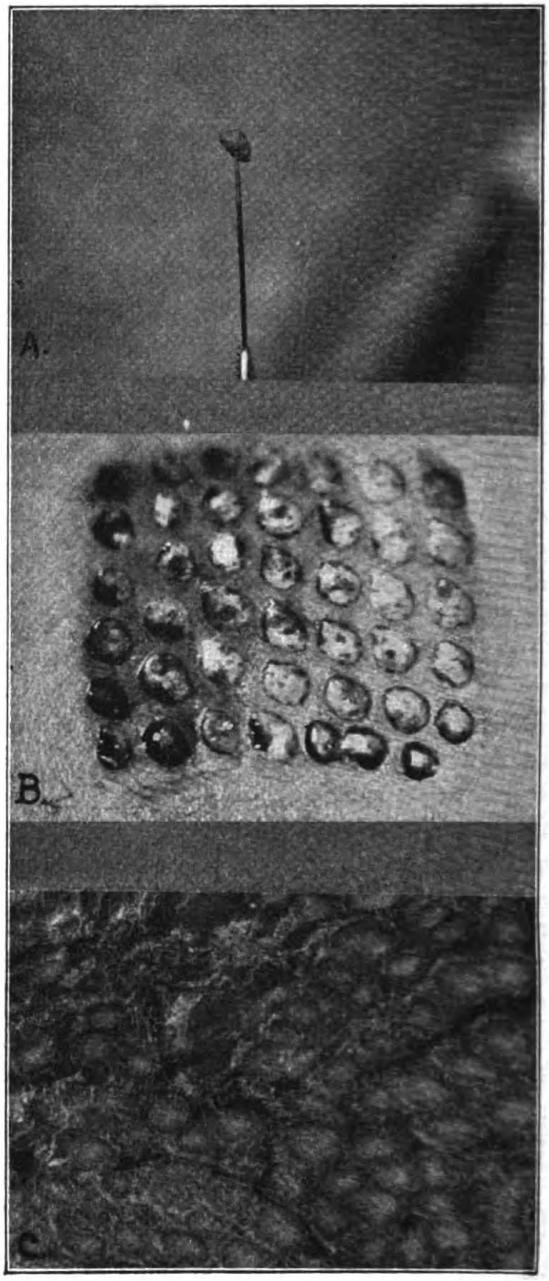

FIG. 3.—A. The actual size of a small deep graft with its edges curled under. When this graft is pressed down on a wound the edges flatten out, thus making the graft about one third larger. B. The punched out defects left by cutting the grafts. Some are filling up with blood in spite of the epinephrin in the local anæsthetic. Note the depth of the pits and the rim of undisturbed skin between them. C. A close view of an area grafted with small deep grafts. The grafts themselves are of normal-appearing skin, which is soft and flexible. Between the grafts is scar-tissue, which is slightly depressed. The scaly appearance of this area is due to desquamation of epithelium. Photograph was taken six months after grafting.

When a large number of these grafts have to be applied the process is very tedious, but this may be mitigated to a certain extent by two men cutting the grafts, while one man places them, or *vice versâ*.

Post-Operative Treatment.

The patient should be kept in bed for a longer or shorter period, depending on the size and location of the grafted area. The part grafted should be immobilised and protected from injury. The dressings should be removed on the second or third day, and any wound secretions carefully mopped up, or washed off with a gentle irrigation of normal salt solution. It is possible, even as soon as this, to tell whether or not the grafts have been successful, and in many instances a narrow ring of new epithelial growth can be seen surrounding each graft. Some bland ointment on old linen is then applied, and if after a day or two the epithelial growth from the grafts is not as vigorous as desired, it is well to alternate the bland ointment with scarlet red, 8 per cent., in petrolatum or zinc ointment, or to use some other epithelial stimulant.

The surface from which the grafts are taken should be dressed in three or four days if protective is used, and the area dusted with dry powder. If silver-foil is used, it should not be disturbed for ten days, after which time all dressings may be removed.

As soon as the new epithelium has filled the gaps between the grafts and joined the epithelium from the wound edges, the grafted area may be dressed with a dry powder, such as zinc stearate, and exposed to the air. I have not found the open treatment of these grafts just after application to be satisfactory. The grafted area should be protected from injury for several weeks. Gentle massage is advisable after about three weeks, and should be continued until the grafted area is as freely movable as the normal skin around it.

There is a marked desquamation of the grafted surface when it dries, especially between the grafts, which is evidently due to epithelial overproduction, but this scaliness can easily be controlled by the application of olive oil or oil of theobroma (butter of cacao).

Untoward Possibilities.

There are certain untoward possibilities. Contraction may occur. Infection may be carried from the granulating surface to the wounds from which the grafts are cut. It is difficult to convey a graft on a needle, or any other instrument, to a granulating wound without occasionally touching the granulations with the instrument. In order to avoid this chance of contamination, it is best either to use a fresh needle for each graft, or to have several needles, so that one can be "flamed" while the other is in use.

Another disagreeable occurrence is the formation of keloid (Fig. 4). Occasionally a massive keloid growth will form in the grafted area, and the grafts themselves can be seen at the bottom of little depressions in the keloid. This keloid growth would probably occur in these wounds whether they were grafted or not. Again keloid may develop in the scars of the wounds from which the grafts are taken. I find that keloid develops either in the wound itself or in the areas from which the grafts are taken, much more frequently in burns than in large wounds from other causes. Burns caused by acids seem especially prone to be followed by keloid.

Comments.

The age of the patient has apparently little effect on the healing of small grafts. The general health should be carefully looked after and when necessary a vigorous building-up treatment inaugurated. Small grafts will often succeed on granulating surfaces under conditions in which no other type of graft could live. There is almost invariably a marked stimulation of the epithelium of the wound edges following the application of these grafts, even though the grafts be unsuccessful.

The skrinkage of the wound after grafting with small grafts is in some cases quite remarkable. Contraction may occur under a grafted area, but it is not nearly so likely as when grafting is not done. If the grafts are placed close together, contraction may be to a large extent prevented. Healing is very much expedited by the use of the grafts, and the resulting cicatrix is solid and resistant.

Small deep grafts are apparently not replaced by scar tissue, as they can be demonstrated as

FIG. 4.—A. The development of keloid on the thigh in the wounds from which small deep grafts were cut. All of these grafts were cut at one operation. There was no keloid formation around the grafts. Photograph was taken one year later. B. Keloid formation around small deep grafts. The grafts, which are soft and in good condition, can be seen at the bottom of little pits. The development of the keloid around the grafts followed a perfectly flat and apparently normal healing. There was no keloid growth in the scars from which the grafts were cut. Photograph was taken nine months after grafting.

definite areas of normal-appearing skin surrounded by scar tissue even after several years (Fig. 5).

A wound grafted with these small grafts, placed at intervals, has a dotted appearance, and the cosmetic result is not so satisfactory as when either large Ollier-Thiersch or whole thickness grafts are used. For this reason it is better not to use them on the face, unless especially indicated. However, if the grafts are placed close together, so as to touch each other, the appearance of the grafted area is very much improved.

A brownish pigment sometimes appears in the grafts themselves, as well as in the cicatrix between them. The scars left by the removal of these grafts is after a time scarcely noticeable, except in those instances in which pigmentation occurs. Sensibility gradually comes in from the periphery, and not from the underlying tissues.

The procedure is the simplest method of grafting. It requires little preliminary preparation after the granulations are in condition, and, unless the area to be grafted is large, the grafting can be done in the patient's room. Enough autografts can be obtained from a small area to cover a large wound without causing any appreciable injury to a patient already in a serious condition.

Several lives have been saved by the use of these small grafts. Thus a child, æt. 12 years, was severely burned over the pelvis, thighs and legs. She came under my care nearly a year afterwards, during which period she had been repeatedly grafted without success. Her condition was desperate. She was delirious, extremely emaciated and generally depleted by the pain, and by the long-standing suppurating wounds. As a last resort the wounds were grafted with small deep, autografts, which were successful, and within a few days the change in her condition for the better was astonishing. Prompt recovery followed, on account of the condition of the granulations. No other type of autograft could have been successful.

The difference in the ultimate result between small superficial grafts and small deep grafts placed close together is almost as marked as the difference in results between Ollier-Thiersch grafts and whole thickness grafts.

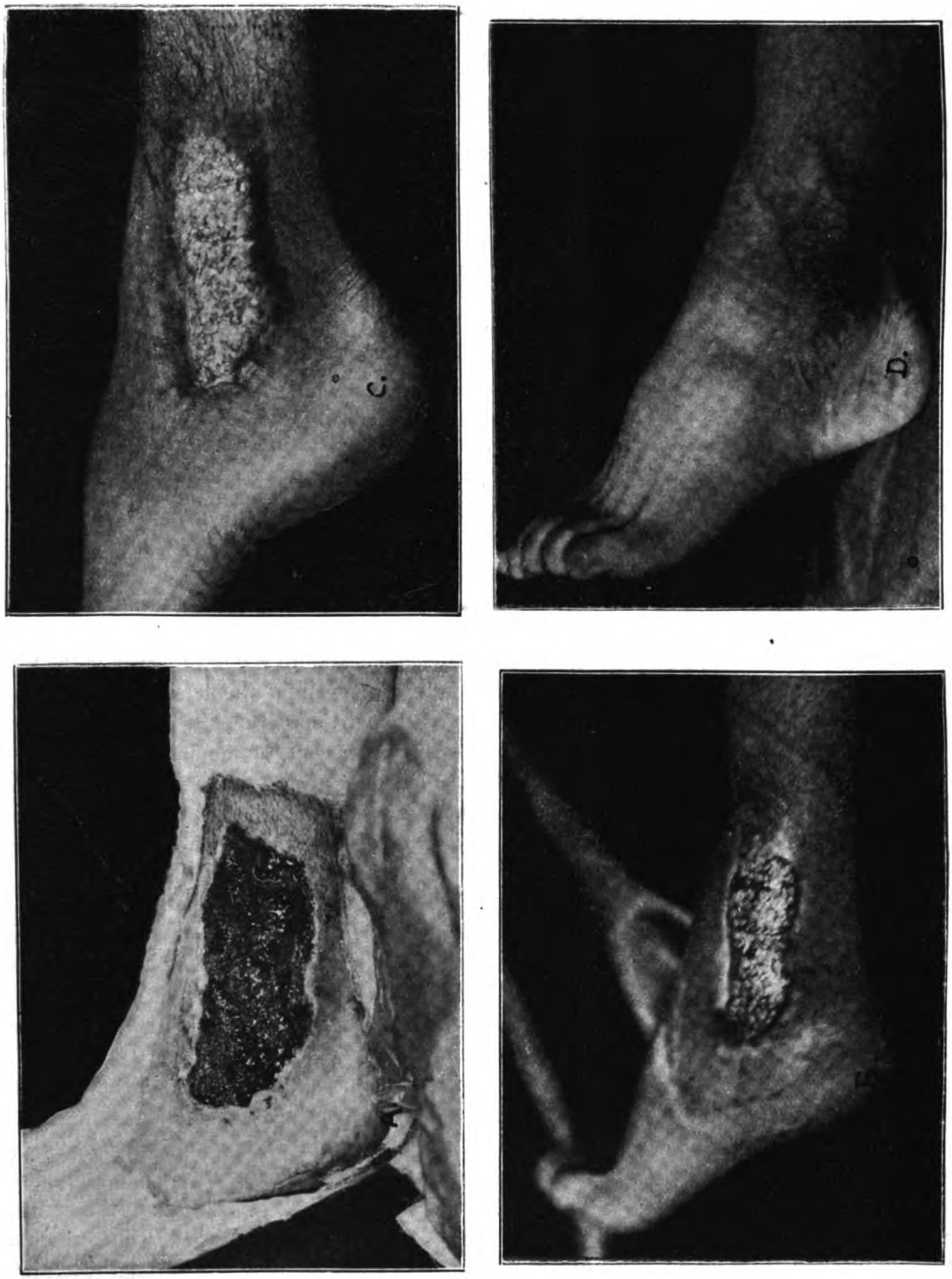

FIG. 5.—A. Ulcer, seven months' duration, following a streptococcus infection. B. One week after grafting with small deep grafts. Note the close proximity of the grafts to each other. Many of them have grown together and also have joined the stimulated skin edges. Shrinkage in the size of the wound is noticeable. C. The same wound two weeks after grafting, entirely healed. D. The grafted area two years and three months after operation. The individual grafts can be plainly seen and are sharply differentiated from the surrounding scar tissue.

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October 4th, 1915.

Treatment of Cancer by Radium.—Prof. Howard A. Kelly states that radium is proving to be of the utmost value in the treatment of cancer of all kinds (including sarcoma), whether operable or inoperable. The beta and gamma rays affect the tissues profoundly, acting much more on the weaker cancer cells than upon the normal cells. Radium therapy is analogous to X-ray therapy, with the striking difference that radium is far more potent and less likely to injure sound tissues. Radium acts more speedily than the X rays. A fibroid tumour may be treated with one or two applications of radium, while many X-ray treatments are necessary, extending over weeks or even months. At first the question was, "Does radium really cure?" This can be answered in the affirmative after the lapse of years. It has also been shown by necropsy made years after radium treatment.

Cancers of the skin are most amenable to radiation. The only obstinate and refractory ones are those which have existed for years, and been treated by cautery, caustics, etc. Those cases are also obstinate which have invaded the deeper tissues, or which have extended through and involved the mucous surfaces. The most obvious advantages of radium are seen in cancers around the eye, ear, lips and nose, where the healing takes place without mutilation, in contrast with operations. Even where there has been extensive ulceration, there is often a surprisingly good restoration of form.

The surgical world has been startled by the rapid results in the hitherto hopeless lympho-sarcomata. Great tumours, interpenetrating all the anatomical structures of the base of the skull, the neck, or the chest, literally melt away in a few days. The rays act like myriads of microscopic knives, attacking every individual invading cell, while sparing normal tissues. Many other sarcomata are also helped in a remarkable manner, but not in such a large percentage of the cases. Epitheliomata of the mouth, tonsils and larynx, especially the basal celled, even in totally inoperable conditions, can be cured in a fair percentage of instances.

A favourable form of cancer is that of the thyroid gland. Thymus growths of the small, round-cell type are often amenable.

Lip cancers yield more and more as our experience grows, but this does not obviate the necessity of a block dissection of the neck.

All breast tumours respond in some measure, the larger medullary form being the most susceptible. The best rule is operation first when feasible and radiation afterwards.

Cancer of the vagina usually disappears rapidly under radiation. Cancers of the cervix are among the most amenable to radium therapy, not only in the early, but even in the later cases where the disease, though massive, still remains localised. In nearly 30 per cent. of the inoperable cases the disease has disappeared under radiation with large amounts (400–600 mgm.) of radium. It is common to witness, too, disappearance of a growth recurring in the vaginal vault after operation. It is a question for the next three years to settle whether we will continue to operate on many of the group of cases which melt down under radium therapy.

Radium has been used repeatedly to shrink an inoperable growth down, and then to operate radically.

To sum up. While radium ought to replace surgery in many instances, and does some of its best work in curing inoperable cases, yet there remains a large place for radium combined with surgery, either by following the surgical operation with radiations to reach nests of cells which the surgeon has not been able to eradicate, or to immunise the tissues, or to remove a growth which shrinks but does not disappear, or to treat recurrences.—*Maryland Med. Journ.*, p. 161.

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We regret that owing to the war the number of pages must be diminished for a time.

EXOPHTHALMIC GOITRE.

By H. J. CAMPBELL, M.D., F.R.C.P.,
Professor of Pharmacology and Therapeutics in the Leeds University; and Senior Physician to the Bradford Royal Infirmary.

IT is an interesting fact—whether a coincidence or a consequence I am unable to state—that in the West Riding of Yorkshire, where the water for domestic purposes is almost exclusively obtained from upland surfaces and is hence very poor in lime, Graves' disease is relatively common, whilst ordinary hard goitre is relatively very rare. This is in marked contra-distinction to what occurs in Derbyshire, parts of Lancashire, and other limestone districts where hard degenerative goitre is common. At the same time, whilst ordinary advanced myxœdema is rare in the West Riding, the modified form which occurs not infrequently as a sequel to exophthalmic goitre, is comparatively common. The natural conclusion to be drawn from these facts is that the exhibition of calcium in cases of Graves' disease occurring in this or similar districts might be expected to produce good results. For the last 15 years or more I have been accustomed to treat all my hospital cases of this disease with calcium in some form and to suggest the same treatment in most of the cases I have seen in consultation. The results have been on the whole good, even when the treatment has not been associated with rest in the open air, or even rest at all, and certainly better than with any other form of medication that I have either myself made use of or have seen in the practice of others. I do not wish to suggest for a moment that the administration of calcium should be the sole treatment applied, but as it is applicable to all varieties of the disease, and to all stages except the very late ones in which the disease is rather due to athyroidea than to an excess of thyroid secretion, it is in my opinion advisable to use it in addition to any other form of treatment that may be adopted. Being purely empirical it

has the advantage of not resting upon theories of causation for its support, theories which in the case of Graves' disease are uncertain and often conflicting and which are still in the condition of being not proven. This method of treatment is also likely to be beneficial in that large class of cases in which the diagnosis of Graves' disease is at least doubtful, and yet where there is enlargement of the thyroid, perhaps slight exophthalmos, occasional tachycardia, and other signs suggestive of over-activity of the thyroid gland. These cases are chiefly met with in young girls who have not long been menstruating, who are anæmic, lethargic rather than excitable, becoming stout instead of wasting, and who, as a rule, quickly improve under calcium associated with iron in some form. Of course, the only cases which are likely to be benefited by calcium are those in which there is a deficiency of calcium in the tissues, but it is on the supposition that there is a deficiency of calcium in the tissues that the explanation of the undoubted benefit that occurs is based. These cases are allied to those which occur after infectious diseases, in which the first symptoms are those of insufficiency of the thyroid secretion, later to be followed by active hypertrophy of the gland with corresponding signs of over-activity. Occasionally both these classes ultimately become cases of true Graves' disease, but the large majority of them never so develop. In true Graves' disease there is always evidence of some abnormal nerve condition; the affection is especially met with in neurotic families or in those who have a neurotic inheritance, as is shown by the occurrence of such diseases as insanity, hysteria, diabetes, epilepsy, or nervous chorea amongst other members, and it not infrequently starts soon after some serious nervous shock. It is far more common amongst women than men. If the best results are to be obtained from treatment, it is imperative that due attention should be paid to the management of the nerve symptoms which form its most pronounced concomitant.

The main symptoms are, exophthalmos with the associated retraction of the eyelids, absence of winking, and insufficiency of convergence on looking at near objects. The exophthalmos is almost always bilateral, though rarely it is noticed first on the left side, and occasionally there is œdema of one eyelid. The exophthalmos is accompanied by enlargement of the thyroid of the soft variety in almost all cases, though at times this is only slight in amount. With the enlargement there is excessive blood-supply to the gland and undue rapidity of circulation through it, as evidenced by the occurrence in it of systolic and diastolic murmurs. Persistent tachycardia is a general symptom, and this may be accompanied by various murmurs heard over either the base or the apex and over the large arteries, due to the rapidity of the blood flow through them. There is not infrequently a galloping or triple rhythm, heard probably all over the cardiac area, the middle sound being accentuated at the apex and the third over the great vessels. Actual valvular disease is rare, though it does occasionally occur. Lacrymation is not infrequently a troublesome symptom, but, of course, only in those cases in which the exophthalmos is considerable. Optic atrophy has been recorded, but it is certainly very rare. Excessive sweating is an almost invariable symptom, and it is usually of the neurotic type, breaking out with excitement and often being limited to the palms of the hands or the upper part of the body. The mental condition is hardly ever normal, there being, as a rule, restlessness, irritability, great excitability, often forgetfulness, and occasionally acute mania or maniacal delirium. Tremors of the hands may be absent in the very early stages, but are pronounced as soon as the disease has become established. Nutrition is always affected, the wasting being sometimes extreme, due to the increased metabolism resulting from, or at least associated with, a marked increase in the oxygen intake. The tremors, though as a rule limited to the extremities, may spread to the whole body. They are fine, regular, and consist of from 8 to 10 oscillations a second. They are increased by excitement and by movement. A dirty discoloration of the skin such as is met with in starvation is frequently present, and other trophic disturbances, such as falling out of the hair, ribbing of the nails, articular swellings, scleroderma, and circumscribed neurotic œdema, may all be occasionally seen. Glycosuria is sometimes met with. Polyuria and increased secretion of saliva are, as one might expect, not uncommon. Diarrhœa, especially of the nervous sort, is rarely absent throughout the course of the disease, but it is generally a somewhat late symptom.

The prognosis is exceedingly uncertain; some cases which, when they first come under observation, appear to be rapidly going down hill, in a surprising way improve, whilst others, which at first appear to be slight, obstinately refuse to yield to treatment. On the one hand, a long continuance of symptoms and, on the other, a very rapidly developing course are unfavourable indications. Emaciation, much pigmentation, and marked mental symptoms are all of unfavourable import. As in so many diseases of a more or less chronic nature, the length of the patient's purse is an important factor in the prognosis, for it is only those who are fairly well off who can afford the prolonged rest under favourable conditions that is advisable, whatever be the stage of the disease, or the indications for treatment in other respects.

Before briefly discussing different methods of treatment, in addition to the administration of calcium, I may shortly refer to methods of causation. Is it primarily a disease of the vagus, of the medulla, in the region of the vagal nucleus, of the sympathetic, or of the thyroid gland, and how far are the symptoms the result of the hyperactivity of the suprarenal glands from the action of an excess of thyroid secretion upon them, and how far are they due to the thyroid secretion itself? Some of the conditions met with in the malady may be the result of overaction of the suprarenals, but the main condition is certainly due to the effect produced by excessive thyroid secretion itself, acting upon subjects of a neurotic type. Many observers consider that the medulla contains a centre, which, when stimulated by thyroid secretion produces tachycardia and exophthalmos, and that, in patients with a neurotic tendency, this centre is more easily excited than in those of a more phlegmatic temperament. It is pretty generally allowed now that the condition is not entirely or even mainly due to an affection of the sympathetic nervous system, for in the majority of the cases that have been examined post mortem, the sympathetic has been found to be healthy; still it must be acknowledged that functional trouble in the sympathetic would satisfactorily account for many of the vaso-motor and secretory disturbances that occur. The parathyroids have also been credited with being the cause of the disease, but it has been pretty conclusively shown that this view cannot be maintained. The balance of evidence is certainly in favour of the opinion that changes occurring in the thyroid gland, resulting in the increase of the gland secretion, are at least the primal causal factor in the production of the disease. Plummer, for instance, after an examination of 3207 cases histologically, states that in every case of true Graves' disease, hyperplastic changes in the gland were present. With regard to the age incidence of the disease, whilst it may occur at any age, the period during which it is most likely to appear is between 20 and 30, that is during the time when the gland normally is tending to show some signs of degeneration, the special demands that are made upon it during the period of active growth having been satisfied. It is true that Filehne produced symptoms of Graves' disease by section of the restiform bodies in animals, and that bulbar symptoms are occasionally met with in the course of the malady, but it yet remains that the only certain method of inducing the assemblage of symptoms, which are in any way characteristic, is by the administration of thyroid juice. Again, whilst excision of the gland in man and in most animals, if they survive the operation for a sufficiently long time for the symptoms to become manifest, results in the appearance of a state which can be neutralised by the administration of thyroid juice, the same condition not infrequently occurs as a sequel to Graves' disease, when over-activity of the gland has given way to atrophy and consequent diminution of activity. If it be allowed that the thyroid hypertrophy, that is to say, the hyperactivity of the gland with a resulting increase in the amount of the thyroid secretion, is the primary cause of the increased metabolism and consequent wasting, the polyuria, palpitation, giddiness, and glycosuria, then some of the other symptoms may be accounted for by irritation of the vagus, either of its nucleus or of its branches reflexly through the connections of the recurrent laryngeal and external laryngeal nerves with the middle cervical ganglion, and of this ganglion with the thyroid branches of the sympathetic in the gland itself. These latter nerves contain powerful vaso-dilators, and their stimulation may result in a lowering of the blood-pressure in the carotids, and may so produce giddiness. Cyon has further shown that they are very readily called into action by the excitation of the cut ends of the vagi, of the de-

pressors, or of the cardiac branches of the recurrent laryngeals. Another evidence of the close connection between the thyroid manifestations and the vagi is the occurrence of attacks of persistent vomiting, which are apt to occur from time to time during the course of the disease.

When the malady occurs in children it is, as a rule, acute, rising to its maximum in the course of a few days, and disappearing in a few weeks. It is instructive that in children, though the majority of cases present in brief all the symptoms met with in the adult, recapitulating in a few days what as a rule in older persons takes years, the majority of patients recover. In isolated instances, however, there is a consequential atrophy of the gland, with a resulting development of the cachexia strumipriva. May not this, in fact, be the true history of the majority of cases of cretinism—an acute attack of Graves' disease in infancy or early childhood, a following atrophy of the gland, and a resulting interference with growth and general development? If this be the case, then it forms a close parallel to the occasional rapid atrophy of the testes after orchitis following on mumps. Given the tendency to the disease the actual causal origin is uncertain. In all probability it may originate from many diverse causes. Bacterial toxæmia, either from water, as in MacCarrison's Gilgit Valley cases, or from pyorrhœa alveolaris, is probably a common cause, whilst undoubtedly, especially in women of a neurotic type, a nervous shock is sometimes followed by an enlargement of the thyroid, and a subsequent development of the disease in an established form.

That treatment is not satisfactory is evident from the multitude of the remedies which have been from time to time advocated for its cure. One reason for the failure that is so frequently met with from some line of treatment, whilst success may follow a directly opposite method, is, I cannot but think, due to the assemblage under one name of cases which differ essentially in their pathology. For instance, if all the cases which show some enlargement of the thyroid, occasional palpitation, and perhaps slight exophthalmos are to be classed as cases of Graves' disease, then I think we are bound to flounder hopelessly in treatment, for the large majority of cases showing these symptoms in a mild degree are merely due to blood-pressure changes, associated with anæmia, as the result of or in conjunction with the onset of menstruation. Again, all cases of hard goitre in which there is deficiency in thyroid secretion, associated with an interstitial cirrhosis, must be excluded, even though they show some slight exophthalmos, for in them the rational treatment is either the administration of thyroid juice or of iodine in some form, the latter being a powerful excitant to the secreting cells of the gland. These cases account, I think, for the successful results attributed to potassium iodide or thyroid feeding in certain cases which have some of the attributes of Graves' disease. It is, of course, in the late stages of Graves' disease, when hypertrophy is being succeeded by atrophy and over- by under-activity, that this line of treatment is the right one. These latter are no longer, however, cases of Graves' disease, but of early myxœdema. Limiting oneself, then, to cases of true Graves' disease, I might first mention various forms of treatment which have been used, but which have given rise to very contradictory reports as to their permanent efficacy. X-ray treatment has been credited with having cured a large number of cases, and the immediate results are often very striking, but as to permanent cure the reports are conflicting. This may be accounted for by the fact that failure certainly followed this method of treatment in many of the early cases, but recent reports with improved technique have given very encouraging results. It must, however, be borne in mind that occasionally disastrous skin changes may occur many months after the treatment has been discontinued. The serum treatment is based upon the view that normally the thyroid secretion acts as an antidote to certain autotoxins developed elsewhere in the body, and, conversely, that these autotoxins serve to neutralise the thyroid secretion. If this hypothesis, and it is only a hypothesis, be true, then when the thyroid secretion is present in excess it is only necessary to inject autotoxins obtained from thyroidectomised animals to neutralise this excess and so to again establish equilibrium. The method has been strongly advocated either in the form of an alcoholic ether precipitate of the milk of thyroidectomised goats (Rodagen), as antithyroid serum, or under the names of antithyroidin and thyroidectin, the latter substance being prepared from the blood or serum of animals whose thyroids have been removed. I have not personally used any of

these preparations to any large extent because the results of the cases in which I have employed them have not been encouraging. Many surgeons strongly advocate complete or partial removal of the gland, stating that even in cases of complete removal there is always sufficient thyroid or parathyroid substance left to prevent the occurrence of myxœdema, and that even if this latter condition should supervene it is a much less serious and more manageable disease than is Graves' disease. Tying the thyroid arteries is a theoretically better procedure, but it has not many advocates. Kocher records out of 59 cases of partial or complete thyroidectomy 76 per cent. of cures, improvement in 14 per cent., slight improveprovement in 3·3 per cent., and death in 6·7 per cent. Most other operators have had a higher mortality, though Dreyfus collected the statistics of 127 cases in which the results were almost identical with those of Kocher. Further experience has hardly borne out these favourable results, and Halsted has recently advocated thymectomy in addition to thyroidectomy as being more beneficial than the latter operation alone.

Personally I have had good results with rest, fresh air, a diet rich in fats and poor in nitrogenous constituents, application of cold locally, the administration of bromide in full doses as occasion required, and the regular use of calcium. Meat juice should be prohibited and milk should be taken sparingly, as both of these act as excitants to the thyroid gland. Belladonna applied locally has seemed to be of use in some cases, but internally I think it is not to be recommended, as it certainly sometimes aggravates the mental excitability. Digitalis is of value when the heart condition indicates the need for its use, but, like many other drugs that have been recommended, it is required not for the disease but for its effects. Local electrical treatment has been advocated, but whilst the good derived from it is doubtful, it certainly in some cases does harm. It is useless, whatever the treatment adopted, to expect rapid results, and in many, in spite of all treatment, the psychic symptoms develop, the emaciation increases, and the end comes either from some intercurrent malady, such as pneumonia, or with startling suddenness from heart collapse. In cases of pregnancy, where the mental loss of equilibrium is often associated with and probably to a large extent caused by the hyperactivity of the thyroid, improvement of the thyroid condition is accompanied by a corresponding improvement in the mental affection. In forecasting the future in a case of Graves' disease it is important to bear in mind that in many cases the disease occurs in those of a markedly neurotic or degenerate type, and that, altogether apart from the thyroid disease, the case may be mentally going downhill. Bearing this in mind I think that, given opportunity for efficient treatment, the prognosis, though always uncertain, is by no means as bad as is often stated, and that recovery, complete and lasting, occurs in a considerable proportion of cases.

October 11th, 1915.

Hæmorrhagic Varicella.—D. G. Campbell reports the case of a girl, æt. 5 months, an inmate of the Montreal Foundling Hospital, who contracted varicella during an outbreak in the institution. On February 25th she showed a few spots, and two days later the temperature rose to 102·4° F., but fell to normal in less than 24 hours. On March 2nd the temperature again rose suddenly to 102° F., and remained elevated till just before death. On March 3rd, at 9 a.m., the eruption became hæmorrhagic, the pustules forming the centre of small purple areas one-fourth inch in diameter. At the same time there was a slight vaginal discharge of blood, and, at intervals, there was epistaxis. At 9 p.m. a sharp hæmorrhage occurred from both mouth and nose, fully 6 oz. being lost. Various measures were tried to check the hæmorrhage with some temporary benefit. The baby took no food, and there was slight epistaxis most of the time. At 9 a.m. on March 4th, a normal stool was accompanied by a good deal of tarry blood, but no fresh hæmorrhage. She died at 11.30 a.m., less than 27 hours from the onset of hæmorrhagic symptoms.

At the necropsy the skin showed hæmorrhagic lesions varying from small points to those about the size observed in varicella. The latter ranged from 2 mm. to 1 cm. in diameter, and were deep red, rather firm, and contained blood-tinged fluid. A few of the larger ones showed slight umbilication. There were peritoneal, pleural and pericardial petechiæ. Hæmorrhagic varicella is rare and usually is fatal.—*Canadian Med. Assoc. Journ.*, p. 610.

NOTES ON RENAL CALCULI.

By GEORGE F. ALDOUS, F.R.C.S.Edin.,
Surgeon S. Devon and E. Cornwall Hospital.

CASE 1.—E. W—, æt. 64 years, seen in consultation with Dr. M. Aikman in March last. She had complained for many years of pain in the left kidney region, and frequently noticed that the urine was thick, had never passed any blood. She was in an extremely nervous condition, which I attributed to years of pain and repeated attacks of renal colic. She was very averse from operation.

On examination a large swelling was felt occupying the left kidney region, extending forwards to nearly the middle line; fluctuation was marked. The diagnosis was a large pyo-nephrosis and a stone. On the following day I operated. On opening the left lumbar region six pints of fœtid purulent urine escaped; the incision was enlarged, and on introducing the hand a hard fixed mass was felt. I was in doubt as to nature of this fixed mass; on viewing it through a Fergusson's speculum it appeared to be a stone. The next difficulty was how to get it out; it yielded, however, to manipulations and was extracted whole. The kidney was represented by a thin-walled capsule, no cortex being left. A large tube was sutured to the skin, and the muscles were united with catgut and the skin sutured with fishing gut, with the tube at the posterior end of wound.

All pain and nervous symptoms disappeared.

The illustration shows the actual size of the stone removed, which weighed 16 oz. and measured $5\frac{1}{2}$ in. long and was 8 in. in circumference.

CASE 2.—J. W—, æt. 24 years, miner, had for many years suffered from pain in the lumbar region and was treated for lumbago, but with no relief. He was admitted to the South Devon and East Cornwall Hospital in March, 1912. Both kidneys were X-rayed and showed a large calculus lying in the pelvis of the right kidney.

This was exposed by the lumbar-iliac incision; the kidney was very large and a stone was felt lying in the pelvis of the kidney. An incision was made

CASE 1.—Renal calculus (actual size).

in the posterior wall of the renal pelvis and the stone extracted. The incision was closed with two layers of suture, and a gauze drain passed down.

The stone weighed 1 oz.

The composition of renal calculi may be due to primary causes or to an infective condition. A primary stone is caused by a deposit of some ingredients of the urine, and is usually composed of uric acid, lime (carbonate or oxalate), or the urates of ammonium or sodium.

An infective calculus is nearly always composed of phosphate of lime and often follows injury, pyelitis, etc. A primary calculus is often coated with phosphates, and may have an oxalate or uric acid stone for a nucleus, pointing to renal infection. The stone in Case 1 is largely phosphate of lime.

The common place for a stone is in the pelvis of the kidney, and in these cases it is usually possible to incise the pelvis to extract the stone and then close the incision with careful suturing as in Case 2. It is sometimes difficult to locate a stone in the cortex, and the surgeon is compelled to incise the kidney along the convex border in order to find and extract the stone; when this has been done it is important not to rely only on the sutures along the convex border. Two or three mattress sutures should be put through the cortex, and the sutures along the convex border should be continued rather beyond the incision made. Since adopting this method I have had no trouble with post-operative hæmorrhage. Renal calculi vary largely in size, from a few grains to several ounces. The largest in the museum of the Royal College of Surgeons weighs 6½ oz.; Shields records one of 19 oz., Barrow one of 16 oz., and Fenwick one of 24 oz., but such large stones are rare.

When formation takes place in the calices the stones may assume a branched shape, united at the base which lies in the pelvis. Radiography is most helpful in the diagnosis of renal calculus, particularly in those cases in which the pain is bilateral as in Case 2.

October 11th, 1915.

IODIDE OF SILVER IN OCULAR THERAPEUTICS.

By PATRICK MOLONEY, L.R.C.P., L.R.C.S.Ed.

As a general practitioner it has often appeared odd to me that there should be so few drugs which can be locally applied to the eyes for, say, the cure of corneal opacities and conjunctivitis, both in its angular and other forms, and for the treatment of gonococcal ophthalmia. Protargol, it is true, is a useful but expensive agent. The ideal therapeutic agent in ocular troubles must be highly antiseptic, astringent, non-irritating, alterative and rapid in action. Iodide of silver, which is inexpensive and easily made, possesses all these qualities, and has given me highly satisfactory results.

The old method of rubbing in the oxide of mercury in the hope of removing opacities in the cornea is certainly slow and wearisome, and requires much labour and patience; besides it is highly irritating. The cornea has no lymphatics, and the oxide of mercury is not a soluble salt, and consequently the amount absorbed must be small.

From the consideration of pathological, histological, and anatomical facts as regards the cornea, it will be obvious that the efficacy of the Ung. Hyd. Flav. is mostly due to its mechanical action. Hence the value and usefulness of the iodide of silver will be understood.

Being troubled with a corneal nebula I perseveringly used oxide of mercury ointment for some weeks, producing irritation of the lids and little benefit. I cast about for an efficacious remedy. Iodide of potassium would, of course, have been the drug were it not too irritating to a mucous membrane. It occurred to me to use a salt which combined the activity of iodine with the astringent and antiseptic qualities of silver. I tried iodide of silver, and to my intense surprise vision began to improve after the first 24 hours. Eventually I showed the eye to a specialist, and he said that the nebula had got very thin.

In the use of this salt, as in the case of most ocular astringents, there are certain contra-indications which must be observed to prevent disastrous results. In distinction from some patent German

salts there is no danger of argyriasis, nor does it stain the skin as the nitrate of silver does.

I always begin with a solution of 5 grs. to the ounce. In gonorrhœal ophthalmia it is most efficacious. One drop of a weak solution instilled 3 times a day or oftener in cases of extensive chemosis and danger of corneal sloughing alters the condition in a few days. Again, in cases of chronic palpebral conjunctivitis, in which a weak perchloride of mercury solution is indicated, the iodide of silver acts quickly. I had also satisfactory results in the treatment of pannus with this salt; in ulcer of the cornea it should be cautiously used, as my experience on this point is not sufficient for me to make a positive statement. All I can say it promises to be of use.

As bacteria play an important part in the diseases of the conjunctiva and a bacteriological examination is made before cataract extraction, the utility of an instillation of this salt will be obvious, especially as its action on the gonococcus is so potent.

The preparation of the silver iodide requires care. I have given Mr. Martindale, 10, New Cavendish Street, W., full details so as to avoid disappointing results.

October 11th, 1915.

Obscure Symptoms due to Accidental Vaccination.—R. Forsius reports the case of an infant, æt. 5 weeks, brought to hospital by the mother, who said that fever, vomiting, diarrhœa, and sores on the mouth had developed a week earlier. The motions had been green and mucous; and the vomiting had usually occurred immediately after a breast feed. After the diarrhœa had lasted about 2 days, during which there were 3 to 5 motions a day, constipation had supervened, and had continued for several days. Though the vomiting had ceased, the sores on the mouth had grown steadily worse, and had prevented suckling. White spots had appeared on the lips and in the mouth, and the mother consequently suspected diphtheria.

The child was well developed and nourished, though somewhat flabby. A thick, greyish-yellow deposit extended practically over the whole of the upper lip, including the mucous membrane of the frontal portion of the alveolar process. The right nostril and the lower lip also showed a similar, but slighter deposit. Ulcerating and slightly bleeding areas, of the size of a pea or smaller, were scattered over the hard palate and dorsum of the tongue. The cause of this condition was obscure, till the writer detected a typical vaccine pustule behind the right ear. The submaxillary glands were slightly swollen and very tender. The throat was not involved, but the temperature was 100·8°.

About 8 days earlier, the infant's father and sister had been re-vaccinated. The father had slept in the same bed as the infant, who had been attended by the sister. Neither the father nor the sister had worn any dressings over the arm. The mother had not been re-vaccinated, as she was nursing the infant, who was considered too young to be vaccinated. A few days after the re-vaccination of the father and sister, sores had begun to appear on the infant's mouth, and in a few days the mother's breasts had begun to itch. On examination they had presented large, partially ulcerating, vaccine pustules, distributed over the nipple and the areolar region, 3 having been found on the right breast, and 8 on the left. In spite of this condition the mother had continued to feed the infant from the right breast till 24 hours before she brought it to hospital. It was evident that the infant's hands had become infected by the father or sister, that its mouth had been infected when it sucked its fingers, and that the mother had been accidentally re-vaccinated on the breast by contact with the infant's mouth.

At first sight the prognosis was very doubtful. The gastro-intestinal symptoms suggested the presence of vaccine pustules in the digestive tract; but these symptoms might also be due to the remote effects of the infection, and the constipation might simply be due to inanition. Suckling was difficult and painful, both for mother and infant. But it seemed a pity to deprive the infant of its mother's milk. She was, therefore, advised to withdraw her milk by hand or with a pump, and to give it to the infant 6 times a day with a teaspoon. Although the milk was much contaminated by the pustules on the breast, this mattered little, as the infant was already practically immune. Under this treatment the infant slowly improved. The crusts on the lips gradually fell off, and were replaced by epithelium. The sores on the mother's breasts also cleared up slowly, and she was ultimately able to resume normal breast feeding.—*Finska Läkaresällskapets Handlingar*, p. 770.

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE

ON

ACUTE ABDOMINAL DISEASES.

By HAROLD UPCOTT, F.R.C.S.,
Assistant Surgeon to the Hull Royal Infirmary; Captain (Temporary) R.A.M.C.

GENTLEMEN,—It is of the utmost importance that you should immediately recognise the early danger signals of serious disease. I wish therefore to-day to bring to your notice certain points in the early stages of some acute abdominal diseases.

First of all I will take the commonest of all such conditions, acute appendicitis. I wish to direct special attention to the sequence of events which occurs in the appendix during the first three days of an attack, and the relation which these bear to the clinical signs.

A year or two ago I prepared material for a paper on this subject, but, owing to the claims of other business, it was never completed except for the title, which was "Time and the Appendix." This was intended to emphasise the importance of the time factor in estimating the changes present in the appendix, and it expresses in a nutshell my theme to-day.

I was led to bring up this subject again by an incident that occurred a few weeks ago. I happened to be operating in a neighbouring town when I got a message from another doctor, who heard I was there, asking me to see a patient of his. He told me that his patient, a young man, had been taken ill with severe abdominal pain and sickness in the middle of the previous night. He was somewhat relieved by morphia, but when seen in the morning the pain had become located in the right iliac fossa, he still vomited and had a slight temperature; in fact, the doctor was able at that visit to make a definite diagnosis of acute appendicitis, and decided to ask me over to see the case

the next day. It was merely owing to the fact that I happened to be near that I was asked to see the case that afternoon.

I found the patient, as his doctor had predicted, suffering from acute appendicitis; immediate arrangements for operation were made and within a couple of hours his appendix was out. It was acutely inflamed with flaccid necrotic patches in its wall, but not yet perforated. As we were looking at this truculent organ, I was led to ask why he was going to wait until next day before sending for a surgeon, and his answer was instructive. He said: "I thought one always waited for twenty-four hours after having diagnosed appendicitis before thinking of operation."

This illustrates a conception of disease which I believe to be wrong; you take a certain group of symptoms which you label appendicitis and for which you have a rule of treatment. Now appendicitis is not a group of symptoms, it is a pathological lesion which produces certain departures from the normal condition of the individual. And surgery enables us to study this lesion in its various stages and in many degrees of intensity, so to account for the differing symptoms that may be produced.

In its beginning appendicitis is a local infection. The mucous membrane of the appendix is lined with a continuous layer of epithelium which forms a barrier between the contents of this most microbic portion of the intestine and the susceptible submucous tissue. The initial lesion is a breach of this layer of epithelium through which bacteria gain access to the deeper parts of the mucous membrane. It is possible to conceive a truly catarrhal inflammation of the appendix, but I have never seen it and so cannot say to what symptoms it may give rise. In practice, however, one uses the term "catarrhal appendicitis" to describe some of the slighter forms of inflammation of the appendix. This little breach in the epithelium, whether caused by the pressure of a concretion, the bite of a threadworm, or the rupture of a lymph-follicle, is the initial lesion. Time and the relative virulence of the bacteria will do the rest.

The beginning of an attack of appendicitis is a local inflammation. This will cause a reflex pain, generally referred to the centre of the abdomen, and reflex vomiting. Since there is absorption of septic material there will be a rise of temperature; this may be only a few points of a degree, but if carefully sought for it will be almost always found, and is, I think, a very valuable sign in differential diagnosis. This septic absorption must pass along the vessels of the meso-appendix, and in a very few hours the nerves of the meso-appendix will be irritated by the spread of the inflammatory exudate, and localised pain is felt, together with deep tenderness and muscular stiffening on pressure.

Here you have the first symptoms of acute appendicitis in order of occurrence:

(1) Generalised pain;
(2) Vomiting;
(3) Local pain and deep tenderness;
(4) Rise of temperature;
(5) Muscular stiffening on pressure.

These five symptoms are not always present, and I would say the most constant are local pain, local tenderness, and temperature. These symptoms will develop within twelve hours from the onset of the illness.

As to the appendix, you will find it stiffened and reddened. Instead of being supple, flexible, and elastic in consistency, it is turgid, swollen, and stiff. The vessels coursing on its surface are distended, and its peritoneal coat is frequently diffusely reddened. Its mesentery is thickened, unusually vascular and often œdematous. On opening the appendix it generally contains a bloody exudate, the mucous membrane is swollen and dusky red, often blotched with sub-mucous hæmorrhages, and it may be ulcerated or necrotic in one or more places.

These are the usual appearances of the appendix removed within twelve hours of the onset of symptoms. It is the condition of the appendix in the stage when it is first possible to make a diagnosis. The inflammation is limited to the appendix—it is an interstitial appendicitis.

What is the further progress of the disease? During the second and third twelve hours there is a gradual extension of inflammation through the wall of the appendix; the peritoneum in its neighbourhood becomes inflamed and produces a slight turbid secretion, followed by a more sticky fibrinous exudate which is the first stage in the formation of adhesions, in which the omentum often plays a part. These are by way of being protective phenomena in readiness for the moment

when the disease ceases to be localised in the appendix. And they are made evident by an increasing stiffness of the muscles and by a more superficial tenderness in the right iliac fossa. During this time the temperature remains elevated or may rise, the pulse-rate begins to increase, and, if the condition is progressing, vomiting may recur.

To recapitulate ; in the first thirty-six hours you have a patient whose illness began suddenly with pain in the middle of the abdomen, soon settling down to the right lower quadrant, he has generally been sick once or twice before he sent for you and may say that he has been feeling chilly. The thermometer shows a slight rise of temperature.

At first the abdomen is soft, but deep pressure in the right iliac fossa evokes a muscular stiffening; later on the abdomen becomes more sensitive, and there is increasing rigidity.

The pulse-rate increases without a corresponding rise of temperature. In children the reaction to the septic absorption as shown by the temperature may be more marked ; in elderly people it is sometimes hardly evident.

Generally there is constipation due to a reflex inhibition of the peristaltic movements of the bowel. Sometimes, however, the disease commences during an attack of diarrhœa. In these cases it is apt to be more rapidly progressive.

This is the stage and these are the symptoms of an interstitial inflammation of the appendix.

The period I have taken of thirty-six hours is purely arbitrary, but it is uncommon to find perforation within thirty-six hours of the onset, while after that time one finds perforation in a small but increasing proportion of appendices.

Operations undertaken after thirty-six hours up to the end of the third day still show in most cases an interstitial inflammation of the appendix, but of increasing severity. The organ is big and dusky crimson, often distended with pus, and frequently shows flaccid, greyish patches in its wall indicating the onset of infective gangrene. A sticky exudate often surrounds the appendix, and beyond this the peritoneal cavity contains a turbid fluid.

As regards symptoms this is the most treacherous stage of appendicitis. The inflammation may be subsiding, in which case *all* the symptoms will diminish. On the other hand, the onset of necrosis generally leads to a lull in the pain which will recur with renewed severity when the necrosis has led to a perforation and the escape of the contents of the appendix into the peritoneal cavity.

Generally speaking, a *continuance* of the symptoms during the third day indicates that the inflammation, though still limited to the appendix, will progress and is unlikely to subside. An *increase* of all the symptoms is of obviously bad import.

A *subsidence of pain*, while the other symptoms remain stationary, is a dangerous sign and suggestive of impending perforation ; likewise, a fall of *temperature* may indicate commencing gangrene, for thrombosis of the vessels in the meso-appendix temporarily blocks the channels of septic absorption. Vomiting often does not recur after the initial sickness, so that its absence cannot be regarded as a favourable sign. It is only a steady diminution of all the symptoms that may safely be taken to indicate recovery from the attack.

Remember, I am speaking only of appendicitis in the first three days. Now consider the further course of the disease. There are three possibilities :

(1) The inflammation may still subside.

(2) Necrosis and perforation may occur and lead to diffuse spreading peritonitis.

(3) Perforation may occur and the infection become localised leading to an abscess.

From the point of view of treatment we may consider these three alternatives in a little more detail.

Firstly, with regard to (1), remember what I have said about appendicitis commencing with a local lesion of the mucous membrane through which the infection spreads to all the coats of the organ. Then you will see that, though the inflammation may subside, healing will only take place with the formation of fibrous tissue, and the appendix will be left more or less crippled and a constant menace to its owner, so that this is an alternative to be avoided.

With regard to alternatives (2) and (3), I need hardly point out that both general peritonitis and local abscess outside the appendix are, to put it mildly, extremely undesirable complications. So we come to the conclusion that all the three possibilities should be prevented. In other words, treatment to cut short the disease, that is operative treatment, must be undertaken before the end of the third day.

We have now to consider: What is the most favourable time in the first three days for operation? I have said that after the first thirty-six hours, though in most cases the inflammation is still limited to the appendix, perforation will be found in an increasing proportion of cases. These cases, then, should be operated on in the first thirty-six hours, for it has been truly said that every case which at operation discloses a perforated appendix has been wrongly treated up to that point.

But this only accounts for a few cases; there remain those in which the disease is progressive or remains unabated, and those in which the inflammation shows signs of subsidence after the first thirty-six hours. As a matter of fact, one cannot tell at the beginning what the event may be, so that rather than wait and risk perforation it is safer to operate on all cases in the first thirty-six hours.

But, why wait for a stated period of time? The case I quoted at the beginning of this lecture was operated on about seventeen hours from the onset of symptoms and nine hours after the diagnosis had been established, and yet the appendix showed necrotic patches covered only by its peritoneum. The diagnosis rested on the pain, vomiting, slight temperature and local rigidity, but the pulse was steady and unquickened and the man did not look ill. There was nothing to show how frail a barrier lay between the septic interior of the appendix and the peritoneal cavity. This case was the exception, but it proved the right of immediate operation. Remember that, other things being equal, an operation for acute appendicitis in the first twenty-four hours is as safe as the interval operation, and in most cases a great deal easier.

So far, I have assumed that you have been able to make a true diagnosis; and in the majority of cases this is possible in the first twelve hours; there is, then, no excuse for further delay in getting rid of the source of the trouble. The difficulty is that often the diagnosis is not such a simple matter; then a reasonable delay is proper until it is evident whether or not the disease requires surgical aid.

As you all know, there are many cases in which the symptoms have scarcely appeared before they fade, often so quickly that you are not justified in diagnosing appendicitis, but at other times unequivocal though fugitive. Such mild attacks are frequently recalled by the medical attendant when a person is suffering from more severe appendicitis.

Such mild but definite attacks should have no successors.

Now there are three small matters worth remembering:

Firstly, do not wait for the appearance of a definite lump in the iliac fossa before making certain of your diagnosis. Such a lump is never seen in the early stages of appendicitis, when the inflammation is limited to that organ. The presence of such a lump means that the appendix has no business inside the abdomen; it is an indication of failure in correct treatment.

Secondly, don't give purgatives in any case suspected of appendicitis. You may reply that slight cases of belly-ache clear up after a dose of castor oil. But they will clear up just as well with starvation. There is nothing more impressive than the constantly recurring history of abdominal pain, constipation, purge, and perforation.

Thirdly, in every case of suspected appendicitis, prop up your patient with pillows into a semi-sitting position.

Then, if your diagnosis is established and perforation should occur before the appendix can be removed, you will have done the best possible thing to limit the spread of the peritonitis.

Since an early diagnosis is of such importance in appendicitis, it is necessary to mention a few of the conditions that may be mistaken for it. There are two extra-abdominal diseases that I have known diagnosed as appendicitis—right-sided pneumonia and right hip disease. The resemblance may be so close that I consider myself fortunate in not having removed the appendix for either condition.

Acute salpingitis on the right side presents a very real difficulty. The sex of the patient, the history of leucorrhœa, and the result of a pelvic examination may render the diagnosis evident. Apart from this the pain of salpingitis is more apt to be localised low in the abdomen from the very beginning, and the early vomiting and fever are less constant. The thing to remember is that the Fallopian tube has a good blood-supply, consequently inflammation is not likely to lead to necrosis and perforation, but generally subsides to a chronic condition, so that no harm is done by

mistaking salpingitis for appendicitis. The contrary error is the one to avoid, for the delay proper to salpingitis may prove fatal if applied to the appendix.

It sometimes happens that the early symptoms of a perforated duodenal ulcer closely mimic the onset of appendicitis. It is hard to put in definite terms the acuteness of pain, but as a general rule the pain of a perforated ulcer is more terrifying in its sudden attack than that of appendicitis, even though it may be felt in the right iliac fossa; and there is more interference with deep respiration. Muscular rigidity is marked and early in duodenal perforation, and early vomiting is less common than in appendicitis. It is important to note that an initial rise of temperature does not occur when a duodenal ulcer perforates.

Another less common condition which may cause confusion is acute inflammation of Meckel's diverticulum. You will remember that Meckel's diverticulum is the unobliterated remnant of the neck of the yolk sac, the omphalo-mesenteric duct. It is found in about 2 per cent. of individuals, generally males, but in only a small proportion of these does it give rise to trouble. It occurs in various degrees of patency, from an impervious cord to a hollow canal like the finger of a glove. It springs from the free margin of the ileum about two feet away from the ileo-cæcal valve, and its other end may be attached to the umbilicus where it pierced the belly wall in fœtal life, or it may have become detached from here and remain free at one end, or its loose extremity may have found a fresh anchorage elsewhere, generally to the mesentery of the ileum.

There are three further points to recollect: firstly, that the incomplete atrophy of the omphalo-mesenteric duct may be associated with malformation of the navel, undue scarring or retraction; secondly, that the process of atrophy may affect the ileum itself, at a point just proximal to the origin of the diverticulum, producing a congenital structure of the ileum; thirdly, the diverticulum may possess a mesentery containing the terminal branch of the superior mesenteric artery, which was originally the vitelline artery, and this fold containing blood-vessels will pass across the ileum to fuse with the mesentery proper. This is all by way of introduction, so that you may realise the pathological possibilities of this remnant of a once useful organ.

The predominant feature of acute diseases caused by Meckel's diverticulum is obstruction. If it is attached at both ends it forms a cord or loop beneath which other coils of small bowels may be ensnared and twisted and strangulated. If one end of the cord is free it may become tied in a knot around a loop of intestine. If the diverticulum is simply a short finger-like process it may be invaginated into the bowel and form the apex of an intussusception. In all these cases the circulation of the diverticulum will be endangered, and it becomes acutely inflamed and ultimately gangrenous.

An accurate diagnosis can rarely be made. If the sudden onset of pain in the lower abdomen followed by early distension and a continued and increasing vomiting with absolute constipation occurs in a boy, there is a possibility of it being obstruction due to Meckel's diverticulum. A history of previous slight attacks of constipation and colic is suggestive, for the pull of a diverticulum will often kink the ileum at its attachment, especially as there is frequently a congenital narrowing of the intestine just above this point. And this may lead to a very important sign—hypertrophy of the intestine above the diverticulum, which may be recognised by the presence of visible peristalsis. Finally, if together with these signs there is some unusual scarring or retraction of the navel, you should think of Meckel's diverticulum.

A cause of acute obstruction less rare than the last, but still fairly uncommon, is volvulus of the pelvic colon. In its acute form it presents a fairly characteristic picture; it generally occurs in men, and the onset is sudden with severe pain and absolute constipation. The pain is increased by rhythmic waves of colic, and the abdomen rapidly becomes distended and drum-like. The patient does not show the early collapse associated with strangulation of the intestine by a band, and vomiting develops late. The symptoms are those of obstruction low in the bowel, plus an amazingly rapid distension which may at first be asymmetrical. This acute volvulus is fairly easily distinguished from acute obstruction due to cancer of the sigmoid, the most common cause of slow intestinal obstruction.

I want to direct your attention to the previous history of these cases of volvulus. You will often find that the patients have suffered repeated minor

attacks of obstruction which have passed off spontaneously or have been relieved by enemata. And after you have noted this fact you will observe these minor cases with closer attention, and you will often find a local swelling in the lower abdomen with visible peristalsis corresponding to the waves of colic. Now this is where the difficulty comes in; for very similar attacks of colic and flatulence with constipation and hardening of a coil of intestine in elderly people form the early signs of a growth of the sigmoid.

I have merely mentioned acute volvulus, a condition which obviously cries for relief, in order to call your attention to the minor cases, and the point to be emphasised is that visible peristalsis means a mechanical obstruction. In all probability relief will be obtained by enemata, especially if given in the knee chest position, and then a very careful examination both through the abdomen and through the rectum should be carried out. If the attacks recur the patient should be examined with the X ray after a bismuth enema.

There is another form of intestinal obstruction with a particular train of symptoms. It is the best example of pure small intestine obstruction without strangulation or other disturbing features—intestinal obstruction by an impacted gall-stone. A calculus must be at least one inch in diameter in order to plug the small intestine; such a stone will have some difficulty in passing down the common bile duct to reach the intestine. It very rarely comes by that route. Stones of this size have generally grown in the gall-bladder, and by their irritation set up a chronic adhesive peritonitis which binds the gall-bladder to some neighbouring organ, by far the most frequently to the duodenum, less often to the colon or stomach. In process of time and ulceration a fistula develops between the two viscera, and the stone is ultimately set free on its downward career.

This early stage in the stone history is a long one and may have lasted many years, and you must remember that one or two large calculi encysted in the gall-bladder do not give rise to what are commonly termed gall-stone attacks—jaundice and colic.

The symptoms of these large stones are less definite, but equally characteristic—a constant dull pain in the right hypochondrium, flatulence and ulness after eating, and much eructation of wind. They have been summed up in the phrase—fair, fat, and forty, and belches gas.

If the stone ulcerates into the stomach it may be vomited; if it is set free into the colon it will be passed per anum; but if it is dislodged into the duodenum, which is by far the most common, it will, if big enough, cause obstruction.

Generally the calculus passes along the small intestine until it finally becomes impacted somewhere in the ileum; this may take several days or a week, and causes a definite sequence of symptoms. While the stone is passing the duodenum and jejunum it causes severe pain in the upper abdomen and profuse and copious vomiting. As it gradually passes down the vomiting becomes less copious, and there are attacks of colicky pain referred to the centre of the abdomen. There may be constipation, but it will not be absolute. The gurgling of increased peristalsis becomes evident, and if the condition has continued for two or three days there may be visible peristalsis. After a variable time when the stone reaches the narrower part of the ileum it becomes firmly impacted and held by the grip of the bowel. Then the constipation becomes absolute, and the vomit begins to turn fæculent. The abdomen is quite soft and only slightly distended. The stone cannot often be felt through the abdomen, for the patients are usually stout, also for the reason that the loop of bowel holding the stone generally drops into the pelvis. At a later stage pressure necrosis of the gut will occur with resulting perforation peritonitis.

Illustrative Cases.

(1) *Tuberculous Disease of Hip mistaken for acute Appendicitis.*

A boy, æt. 8 years, was taken ill with pain in the right leg and upper part of thigh. The next day he vomited and complained of pain on micturition. Two days after the onset he was admitted to the infirmary and was treated in Fowler's position with continuous saline infusion and starvation.

When I saw him two days later the abdomen was tender and rigid, especially in the right iliac fossa. The right thigh was flexed and extremely tender in the groin, where there was some swelling.

During the next few days a lump appeared in the iliac fossa which gradually increased in size.

Exploratory incision just above the outer end of Poupart's ligament revealed an abscess beneath the fascia iliaca, containing white, odourless, creamy pus. Communicating with the back part of this cavity by a constricted opening was a more extensive cavity in the sheath of the psoas. Subsequently a narrow prolongation of the abscess cavity was found passing under Poupart's ligament to the under surface of the neck of the femur. At a later date an excision of the hip was performed, and the boy eventually made a good recovery.

(2) *Obstruction due to kinking over the Mesentery of a Meckel's Diverticulum.*

A boy, æt. 7 years, a patient of Dr. H. L. Evans, of Hull, was seized with pain in the upper abdomen, chiefly above and to the left of the navel. Next morning he was taken to a physician. He had then vomited several times, but was able to walk into the consulting room. His bowels had been opened early that morning. The pulse was 70, and the abdomen full, with distended coils of small intestine visible above the umbilicus. He was sent into a nursing home, where he became rapidly worse, and I was hurriedly summoned at 3.30 in the afternoon. I found him profoundly collapsed, eyes sunken and dark rimmed, pulse hardly to be felt. The vomiting continued. The abdomen was distended, and prominent coils of small intestine were seen across its upper part. No tumour could be felt.

I opened the abdomen half an hour later and found the distension of the intestine ceased at a point in the ileum where a patent Meckel's diverticulum two inches long sprang from it. The diverticulum was quite free and not inflamed. Its mesentery was kinking the bowel at this point. A soft rubber catheter was tied in the diverticulum, which was brought to the surface and fixed in the lower angle of the wound. The boy made a smooth recovery, and a fortnight later I excised the diverticulum.

(3) *Acute Obstruction. Strangulation by obliterated portion of Meckel's Diverticulum.*

I was sent for by Dr. E. B. Morley, of Barton, to see a little girl, æt. $2\frac{1}{2}$ years, who had been taken ill two days previously with pain in the right side of the abdomen. The next day she took practically no food, and the bowels did not act Early in the morning of the third day she began to vomit, and this soon became fæculent. An enema was given, but returned unchanged. In the afternoon she became rapidly worse, and on my arrival in the evening I found the child restless, sunken-eyed, vomiting without effort every few minutes. There was a dusky pallor of the skin, and the pulse was about 200. The abdomen was moderately distended, but soft, and a tender mass was felt to the right of the navel, which seemed fixed to the posterior abdominal wall. There was a slight umbilical hernia. Rectal examination revealed nothing.

In considering the diagnosis intussusception and Meckel's diverticulum were suggested as being possible causes of the obstruction.

After an intravenous saline infusion, the pulse improved slightly in volume, and I opened the abdomen at 8 p.m. The lump was found to be a coil of small bowel tightly snared by a band. In the centre of the loop was a tense tumour, of the size and colour of a plum, with which the band seemed to be connected. After dividing the band, the plum-coloured mass was seen to be a strangulated Meckel's diverticulum springing from the ensnared bowel. It showed commencing gangrene around the neck. The loop of intestine was dusky red and œdematous. The diverticulum was cut off and a rubber tube fixed into the gut through the opening by a purse-string suture.

The child died suddenly after a vomiting attack about two hours later.

(4) *Obstruction due to impacted Gall-stones.*

I was called by Dr. A. L. Tireman, of Hull, to see a woman, æt. 65 years, suffering from abdominal pain and sickness of one week's duration. She had suffered from "bilious attacks" for several weeks, but was taken worse a week ago with general abdominal pain and sickness. Since then the pain had varied in position, but was generally in the neighbourhood of the umbilicus. During the last two days vomiting had been progressive, and there had been absolute constipation.

She was admitted to the infirmary, and at the operation an impacted gall-stone $1\frac{1}{2}$ inches in diameter was removed from the ileum. She made a smooth recovery.

October 18th, 1915.

CLINICAL JOTTINGS.

(*Continued from p.* 312.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

The Treatment of Phthisis by the Production of Pneumothorax.

This is really an old question which has been recently resuscitated. It is based upon the theory that in the compressed lung tubercle ceases to develop, but the theory is erroneous, and therefore the practice based upon it will be unsound.

Of course with modern aseptic methods sterilised air can be introduced into the pleura without much risk, yet if by any accident infection occurred and an empyema resulted, the last condition of the patient would be worse than the first. Yet, assuming all goes well, the question is, What will be the result of the artificial pneumothorax? In the first place the pneumothorax so produced will not be effective long, for the air is readily absorbed and in a few days will entirely disappear. In the second place, if the pneumothorax were maintained, still the progress of the tubercle in the compressed lung would not be arrested. Indeed, owing to the adhesions which have formed over the most affected portions of the lung, the pneumothorax would only affect those portions of the lung in which the mischief was not advanced. But there is another risk of an altogether different kind, of which I have seen a good many instances in spontaneous pneumothorax. As the one lung collapses the contents of the tubercular cavities are forced into the air-tubes, and may easily pass into the other lung, where they may set up acute tubercular aspiration-pneumonia, and lead to a very acute development of tubercle in the other lung. Again, I have seen more than once very urgent, even fatal, dyspnœa produced in this way. It follows, therefore, that though the actual operation of artificial pneumothorax is not specially risky, yet the indirect risks are by no means small, while the prospects of amelioration, still less of arrest, are practically *nil.*

Plastic Bronchitis and Asthma.

Attacks of dyspnœa, paroxysmal in character, and often diagnosed as asthma, occur in the course of plastic bronchitis. Where the casts are large and freely expectorated the attention is arrested and the diagnosis easily made. Where the casts are small the real condition may be overlooked; but if plastic bronchitis be thought of and the sputum examined the nature of the case is obvious. True spasmodic asthma begins in early life. Plastic bronchitis, though met with in children, is more common in adults. Indeed, asthma-like attacks commencing in adult life, are probably not spasmodic asthma at all, but some other form of paroxysmal dyspnœa.

The correct diagnosis of plastic bronchitis is important because it is in most cases easily cured by iodide of potassium.

(*To be continued.*)

Variola Simulating Measles.—T. Hellström reports the case of a stewardess, æt. 40 years, attached to a ship trading between Sweden and Finland, who developed fever, severe headache and general malaise on a Thursday. She became steadily worse till the following Sunday, when the appearance of a rash, most vivid on the face, was accompanied by a sense of well-being. The rash suggested measles, but for safety's sake the patient was sent to an isolation hospital. Here also measles seemed the most probable diagnosis. The whole face was covered with a papular rash, the papules being very large and close together, although distinct from each other. An examination of the mouth showed spots, resembling Koplic's spots, but on further investigation many other spots were found scattered over the mouth and throat. The whole body was covered by a papular rash like that of measles, but on the backs of the hands an eruption began to appear which was characteristic of variola. The subsequent course of the disease proved it to be variola.—*Svenska Läkaresällskapets Förhandlingar*, p. 571.

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We regret that owing to the war the number of pages must be diminished for a time.

LECTURE

ON

SOME POINTS IN THE GENERAL STRUCTURE AND FUNCTIONS OF THE NERVOUS SYSTEM.

By H. CAMPBELL THOMSON, M.D., F.R.C.P.,
Physician to the Department for Nervous Diseases, Middlesex Hospital; Physician to the Hospital for Epilepsy and Paralysis, Maida Vale.

THE brain and spinal cord are composed of three principal elements:

(1) Connective tissue.
(2) Vessels.
(3) Nerve substance.

The connective tissue acts mainly as a support to the nerve tissue and is called neuroglia. The vessels are the channels through which nutrition is obtained and the nerve substance is the matter on which the properties special to the brains and cord depend. This nerve substance, when reduced to its elements, is found to consist of nerve cells and processes, and the view is now generally accepted that every nerve cell and its processes forms a unit which is anatomically independent of every other unit. The fibres from one unit may interlace with those of another, but the life of each is separate, so that the death of one need not necessarily affect the other. This fact may be well understood by comparing a group of neurons to a closely growing group of trees. The branches of the trees interlace with one another so freely that when the leaves are on one might easily imagine them to form a continuous network, but if one tree dies its branches wither without affecting those with which they interlace, and it becomes obvious that while the outgrowths were in contiguity there was no continuity, and that each tree is dependent for its own life on its own nutrition. So it is with the cell body and its processes. Each is independent of

the other, and the processes are dependent for their nutrition on the cell bodies just as are the branches of the tree on its roots. The cell-body and its branches are together called a neuron. The branches conducting impulses towards their cell body are generally those known as dendrons, while those conducting outwards are known as axons. As a general rule, the dendrons are the shorter and more numerous, the axon generally being single and of considerable length, but exceptions to this statement are not infrequent.

The individual neurons are connected together in separate bundles, and different bundles serve different purposes.

Thus there are some bundles which serve the purpose of conducting impulses which lead to movement, others which conduct impulses of sensation, and others again which serve the purpose of associating some of the main bundles with one another.

So far, then, we have seen that the brain and cord consist of bundles of neurons which are supported by connective tissue substance, and the next step is to investigate the manner in which these bundles are arranged.

First, take the tracts which conduct voluntary motor impulses. Here we find two main divisions which are known as the upper and lower motor neurons. The upper neurons have their cell-bodies situated in the cerebral cortex, and their long axons converge till they become collected into a concise bundle which passes through the internal capsule, the crus cerebri, the pons, and finally reaches the medulla. Here the majority of the fibres decussate with their fellows of the opposite side and then form that part of the cord known as the *crossed pyramidal* tract. As the fibres pass down this tract they end at different levels by communicating with the cells of the anterior horns of the cord.

Those fibres which do not cross in the medulla pass down the cord to form the direct pyramidal tracts, the fibres of which decussate in the cord and finally likewise communicate with the cells of the anterior horns. It should be mentioned that the fibres which conduct motor impulses to the face decussate before they reach the medulla, so that they can communicate with the nucleus of the facial nerve which corresponds to the motor cell of an anterior horn of the cord.

We are now in a position to trace the impulses which cause a voluntary movement. These impulses originate as a mental process and are then switched on to the appropriate bundle of neurons whose cell-bodies are, as we have seen, situated in a definite part (the so-called motor area) of the cerebral cortex. Suppose the subject decides to move an arm. The impulse is directed to the particular set of neurons which form the pathway to the arm. It goes down this pathway, passing on its way the anatomical points already mentioned, namely, the internal capsule, the crus and pons, and then reaches the medulla. Here the impulse finds its main pathway crossing to the opposite side and, passing on, it finds its way down the cord along the pathway known as the crossed pyramidal tract until it reaches the level of the nuclei of the nerves going to the arm.

Here the impulse finds that the neuron along which it has been travelling stops, but, by a mechanism the exact nature of which is not fully understood, it is able to pass over to the large nerve cells in the anterior horn of the cord. These, as we have seen, are the starting point of the lower neuron, and the impulse now finds a straight path down the peripheral nerves along which it runs and finally sets in movement the various muscles which it started with the object of doing. If a movement of the face is required the impulse will leave the main path in the pons and communicate with the cell of the corresponding lower motor neuron, which in this case is represented by the nucleus of the facial nerve.

Similarly, to obtain a movement of the leg, the impulse will pass down the cord until it reaches the level of the cells from which the leg nerves arise, when it will pass over to these and then travel down the peripheral nerves to the muscles, as in the case of the arm. If the continuity of this path is interrupted at any point the impulse will be unable to make its way down, and consequently no voluntary movement can take place.

The next point to consider is how we may know at what point in the motor tract the interruption has taken place, and the first step in this respect is to decide whether it is the conductivity of the upper or the lower neuron that is at fault. Fortunately there are generally some striking points of difference by which this can be decided. You will remember that the two motor neurons

are independent of one another anatomically, but intimately related to one another physiologically, inasmuch as impulses are continually passing from the upper to the lower.

Now if there is an interruption in the course of the upper neuron there is, besides some loss of power, also some loss of control which is normally exercised by the upper over the lower neuron. This loss of control is shown by an undue irritability of the isolated lower neuron. Thus the tendon reflexes become exaggerated, as shown by the increased knee-jerks and tendo-Achillis jerks. There is also often a continued clonic contraction of the calf muscles produced by sharply flexing the ankle, a condition known as ankle clonus. The muscles tend to become rigid or spastic, and since rigidity is not equally balanced certain contractures take place in which, in severe cases of cord diseases, the limbs tend to assume a position depending on the predominance of the flexors and adductors over the extensors and abductors. Another and most important point for diagnostic purposes is the behaviour of the plantar reflex. When the sole of the foot is stimulated in the case of an infant the movement of the big toe (which is the essential movement of the reflex) takes place in an upward direction, *i. e.* the toe is extended and gives rise to what is generally spoken of as an "extensor response." About the time the infant is thinking of beginning to walk it will be noted that on stimulation of the sole the extension of the toe has given place to flexion. The toe now bends downwards instead of upwards, and is said to have a "flexor response." This flexor response persists during the remainder of life unless any interruption of an organic nature takes place in the conductivity of the upper motor neuron, when the response again assumes the extensor form of the infant. Therefore from the clinical point of view *an extensor response indicates a lesion of the upper motor neuron.* This is often called Babinski's sign, after the French neurologist who first demonstrated its significance.

Thus as effects of an upper motor neuron lesion we have mainly:

(1) Exaggeration of tendon reflexes.

(2) Rigidity and contractures.

(3) Extensor response of plantar reflexes.

On the other hand, if the continuity of the lower motor neuron is interrupted we find signs of failing nutrition in the muscles, since these depend for their vitality upon the cell-bodies in the anterior horns.

Thus one of the most prominent signs of disease of the lower motor neuron is wasting of the muscles, and this loss of nutrition can be further demonstrated by alterations in the way the muscles react to electrical currents. The reflexes of the part affected will be diminished or lost since the reflex arc on which they depend for their existence is broken, and there will be flaccidity of the limb instead of the rigidity which is so prominent a feature of lesions of the upper neuron. In late stages of lower neuron disease the limbs become deformed through the contraction of the wasted fibres of the paralysed muscles and through the unantagonised action of these that still possess any power.

A simple example of an upper neuron lesion is to be found in a case of hemiplegia, such as may occur after a hæmorrhage in the neighbourhood of the internal capsule. In such an instance the conductivity of the main motor tract is interrupted, so that impulses for voluntary movements can no longer pass through, with the result that the patient has some loss of power in the opposite side of the body.

The effect is, however, not confined to the mere loss of power, because as soon as the impulses cease to pass, those of other tracts become over active, and so, in addition to the negative result, there are also signs of over activity, which lead to rigidity and contractures.

Normally, the act of moving is accomplished as the result of a collection of influences which all work in unison, and when, for any reason, one out of the set is nullified, the balance of power is upset, with resulting disorder of movement of one kind or another.

When, for instance, the main motor tract is interrupted, as described above, the balance of power between the cerebrum and the cerebellum is upset, with the result that the cerebellar influences tend to predominate and to set up rigidity of the affected limbs. The arm tends to be drawn over the chest in a flexed position, while the leg is stiffened by the over action of the extensors of the knee-joint, and the patient has difficulty in bending it, and walks as though he had a wooden leg. In other cases the predominant influences cause involuntary movements such as athetosis and tremors.

It must therefore be borne in mind that the results of an upper motor neuron lesion are two-fold in character.

(1) *Negative.*—Due to loss of function of the injured tract, which results in loss of voluntary power to carry out certain movements.

(2) *Positive.*—Due to over action of other tracts, which results in such conditions as rigidity, tremors, and athetosis.

On the other hand, when the lower motor neuron is injured from the cells of the anterior horn outwards, the influences from all the upper tracts which have converged upon it are annulled, and the result is a flaccid paralysis followed by wasting of the muscles and changes in their electrical reactions. Later on there may be deformities, but these are more of a mechanical character and are not due to the influence of other tracts, since no such influence can get through the single path of the lower neuron if its continuity is destroyed.

A good example of this type of paralysis is to be found in acute poliomyelitis, commonly known as infantile paralysis. Here the lower motor neuron is affected at the cells of the anterior cornua, and consequently those nerve fibres and other structures which depend on the integrity of these cells for their nutrition undergo degenerative changes. Thus a limb affected in this way becomes smaller and colder than its fellow, and is apt to be the seat of ulcers, which are often very troublesome to cure, and as time goes on, unless great care is taken, the different parts of the limb assume faulty positions, in which they are very likely to become fixed. The treatment of lower motor neuron lesions of this kind consists largely in trying to improve nutrition by massage and electricity, in correcting the tendency to form faulty positions, and in preventing over-stretching of paralysed muscles.

October 25th, 1915.

CARCINOMATA OF THE COLON, WITH SPECIAL REFERENCE TO THE TRANSVERSE COLON.

By EDRED M. CORNER, M.C., F.R.C.S.,
Surgeon and Lecturer to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond Street.

No surgeon has an extensive experience of tumours of the transverse colon, on account of their relative infrequency, and because in the great majority of instances the surgeon is operating to relieve intestinal obstruction primarily, and the finding the carcinoma secondarily. The relative importance is shown in the figures of the incidence of carcinomata in different parts of the abdominal colon, *i. e.* exclusive of the rectum. The figures have been gleaned from the records of St. Thomas's Hospital as contained in the St. Thomas's Hospital Reports for 1900–1911 inclusive.

	Per cent
Carcinoma of the cæcum	20
,, ,, ascending colon	10
,, ,, hepatic flexure of the colon	4
,, ,, transverse colon	18
,, ,, splenic flexure of the colon	2
,, ,, descending colon	6
,, ,, iliac and pelvic colon	40

Carcinomata of the colon are most frequent in the iliac and pelvic colon, then in the cæcum, and then in the transverse colon. In all other situations they are relatively infrequent. Carcinoma is more frequent where the "food-stuffs" remain and pause in their passage through the intestine than where they are in motion. Its incidence is more frequent in the colon on the right (hepatic) side than in the colon on the left, *i. e.* where the bowel contents travel against gravity, and therefore more slowly than when they move with gravity.

Of these carcinomata some remain quiet and produce no symptoms or signs until they are very far advanced. Others cause intestinal obstruction quite early in their course. In consequence carcinomata in the various regions of the alimentary canal possess various relationships to the symptoms which the case presents, such as acute abdominal

disease, acute or chronic intestinal obstruction. In other words, carcinomata in the different regions of the colon show different pathological and clinical characters.

In his most interesting address at Aberdeen, our President,* Sir John Bland-Sutton, said that surgeons tended to be of two kinds—technicians or craftsmen, who improve the art of their profession, and biologists, who improve the science of their profession. It may be added that the value of the work of both has to be brought before and estimated by the clinicians. Hence I would suggest that the greatest and most important group of surgeons is the clinical, as they have to appraise the work of all other departments. To appraise the value of the pathological knowledge of the incidence of carcinomata of the large intestine obtained, as above, from the pathologist an inquiry was made as to their relative importance in causing acute intestinal obstruction.

Relative Importance of Carcinomata of the Colon in causing Acute Intestinal Obstruction.

	Per cent.
Carcinomata of the cæcum	4·1
,, ,, ascending colon	·7
,, ,, hepatic flexure of the colon	2·5
,, ,, transverse colon	13·4
,, ,, splenic flexure of the colon	17·1
,, ,, of the descending colon	5·1
,, ,, iliac and pelvic colon	59·8

This is a very different table from the last, and shows:

(1) Carcinomata of the cæcum, and still more carcinomata of the ascending colon, do not seem to obstruct the intestinal flow. The growths tend to be lateral and local in the gut wall, like carcinomata of the rectum, and hence they do not obstruct the flow within the bowel like does a ring growth.

(2) Carcinomata of the iliac and pelvic colon, splenic flexure and transverse colon are quite different in their clinical behaviour, causing intestinal obstruction early in their course.

(3) Carcinomata of the splenic flexure are infrequent, but practically always cause intestinal obstruction. Carcinomata of the hepatic flexure show the same in a much less marked degree.

As many regard the large intestine as a whole, it may be as well to include amongst the carcinomata those of the rectum in the table of the relative importance of carcinomata of the large intestine to cause acute intestinal obstruction.

	Per cent.
Carcinoma of the cæcum	3·3
,, ,, ascending colon	0·2
,, ,, hepatic flexure of the colon	1·7
,, ,, transverse colon	11·0
,, ,, splenic flexure of the colon	14·3
,, ,, descending colon	4
,, ,, iliac and pelvic colon	47·0
,, ,, rectum	18·5

It is now seen that the asymmetrical and "lumpy," as contrasted with the asymmetrical and annular carcinomata, of the right-sided segment of the colon are the least frequent to cause acute intestinal obstruction. This clinical condition increases in frequency when the growth is in the transverse colon, at the splenic flexure, to reach its maximum clinical frequency in the iliac and pelvic colon, where it takes the form of a ring-like band, from the direction of the lymphatics, constricting the bowel. In the rectum again the growth is "lumpy" and asymmetrical in its disposition.

It will be seen that, looked at from all points of view, carcinomata of the transverse colon occupy both pathologically and, to a lesser degree, clinically an intermediate position between cæcal and sigmoid carcinomata; whilst, in cases of acute intestinal obstruction where the situation of the cause cannot be ascertained, the preponderance of the left-sided causes over those on the right side suggests the very practical point of opening the abdomen through the left rectus muscle.

Clinically carcinomata of the transverse colon exhibit these characteristics: the absence of symptoms warning to the patient, the unannounced onset of acute intestinal obstruction, the presence of a small annular growth. Hence, when it comes to treatment the surgeon has two things to attend to—intestinal obstruction primarily and a carcinoma of the transverse colon secondarily. The former is a condition abounding in micro-organisms. The

* Medical Society of London.

organisms are responsible for many and much of surgical failures in the removal of growths from the colon. Hence, I would urge that the surgeon is very unsound who attempts to do constructive work, *e. g.* the plastic work of an anastomosis, in the face of this sepsis. Therefore, when any degree of acute intestinal obstruction is present a temporary colostomy alone should be done. The artificial anus and the growth are removed about three weeks later, the ends of the resected bowel sutured, and the cæcum anastomosed to the sigmoid as indicated in the figures.

It is noteworthy that it is strongly advised that the surgeon do nothing, not even removal of the growth, but relieve the intestinal obstruction. In the writer's experience patients so treated do better in both their early and late clinical post-operative stages than if the growth is removed and Paul's tubes inserted. It is far better to leave the resection of the growth until the time of the anastomosis. An inflamed growth is far more likely to lead to trouble if any attempt is made to remove it than does an uninflamed growth. This remark applies both to near and remote sequence. Thus

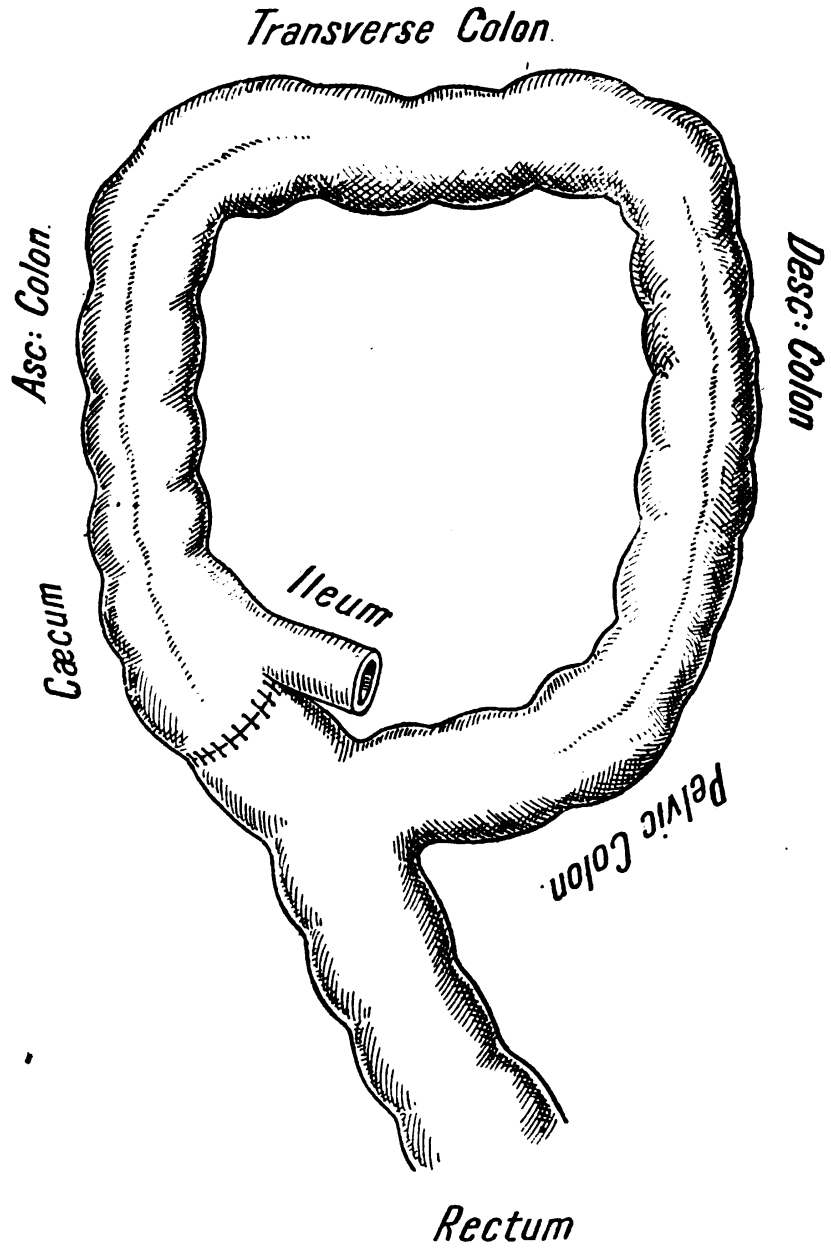

FIG. 1.—Showing loop of large intestine completed by the anastomosis of the sigmoid to the cæcum.

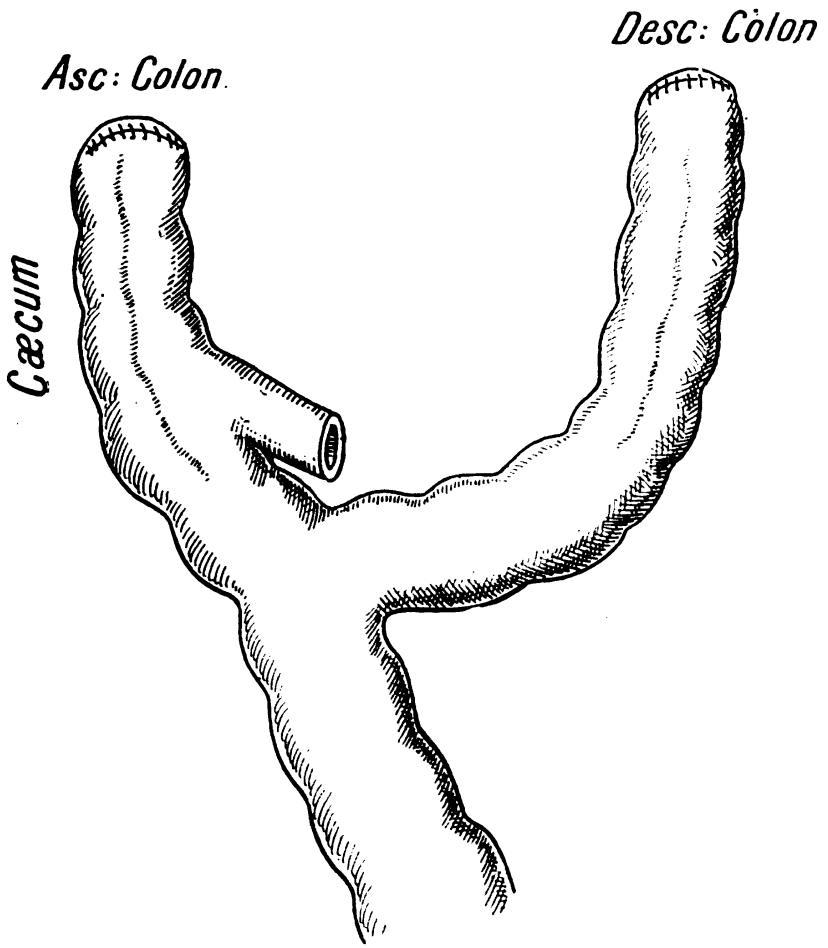

FIG. 2.—Illustrating the anastomosis and the resection of the transverse colon.

I do not like "playing" with inflamed growths any more than I like "playing with fire." The best work is usually done with cool materials.

From time to time carcinomata of the transverse colon are large, and their removal may require a partial resection of the stomach. Or they may become adherent to the umbilicus and discharge through it. But these features are unusual and need not be introduced into a discussion on general principles. In practical surgical work we are far most likely to be called upon to relieve the intestinal obstruction caused by a carcinoma of the colon. Having done this it may have to be decided how to resect this carcinoma. The anastomosis may be made local to the excised carcinoma when possible, and there may be an oblique or circular enterorrhaphy. If, as is the usual case, the excision of the artificial anus and the growth leave too little transverse colon behind to allow of an end-to-end anastomosis being done, I believe the best proceeding is to close the ends of the bowel; cut off the caput coli with the appendix, anastomose by implantation the cæcum to the upper rectum or sigmoid. This anastomosis has the advantages of keeping our second stomach, the cæcum and ascending colon, so that less food escapes absorption than if these structures are removed, the appendix, always abnormal in these cases, is removed, and the anastomosis, though made far from the growth, is yet conservative.

With regard to proceeding to the radical measures in the excision of the growth one's advice would be—Never where possible try radical measures on an inflamed growth. Do not be deterred by the

presence of enlarged glands, as many are inflammatory, but beware of intra-peritoneal adhesions. In other words, the surgeon need not be put off by extra-peritoneal inflammation, but he can be easily stayed by intra-peritoneal inflammation.

There is no doubt about the attempt at the spontaneous cure of the carcinoma by the production of contracting scar tissue by the inflammation of the contiguous structures. It is the contraction of this non-carcinomatous tissue which produces the intestinal obstruction in ring-carcinomata. Hence, ring-carcinomata are more amenable to operation and give better results from operation than do local and lumpy carcinomata. Hence, I would make the statement that the results from the judicious resection of the ring-carcinomata of the transverse, iliac and pelvic colon give better results than do resections for asymmetrical carcinomata of the cæcum and rectum. To demonstrate this it is practically always necessary to do the operation in two stages, and patients do not always like this. Firstly, do a colostomy, relieve any obstruction and make the tissues less septic; secondly, resect the growth, anastomose, closing the colostomy.

In conclusion, I must say that I regard a transverse colostomy as only a temporary measure. Colostomies have in general to be where the disposition of troubles put them. The inguinal colostomy is the one for choice. In general, its results are better, more certain, and its performance easier, and it leaves a longer piece of colon for food absorption. Colostomies are often olfactorily unpleasant, and something must be left to the choice of the patient. The transverse colotomy is apt to be the more obtrusive. And it is a matter of taste whether the patient prefers to have fæces in his umbilicus or in his pubic hairs. But putting aside all æsthetic considerations, on surgical grounds I think the inguinal colostomy better. A transverse colostomy has a secondary and distinct *rôle* in surgery.

October 25th, 1915.

The Uselessness of Strychnine in Heart Failure.—L. H. Newburgh points out that although many clinicians prescribe strychnine in acute and chronic heart failure, because they believe that it increases the work of the heart and that it slows and steadies the pulse, yet there is no sound basis for this belief. Pharmacologists have not been able to demonstrate that strychnine increases the output from the heart. Clinicians have not shown that broken cardiac compensation can be relieved. Sir James Mackenzie, discussing "cardiac tonics," has written: "The most popular remedy of this class is strychnine. I have carefully sought for its special effect on the heart, and found none. The evidence is practically all clinical, and endows the drug with the most diverse properties. It is recommended as a cardiac stimulant in slow hearts, and even in heart-block it is said to quicken the beat. It is also recommended in the too excitable heart, as when there are extra-systoles, and in the rapid heart of acute myocardial affections. It is said to be beneficial in cases of low blood-pressure and equally beneficial in the cases of high tension and even in angina pectoris."

In England Parkinson and Rowlands investigated the action of strychnine in 50 cases of severe heart failure with the polygraph and manometer. Readings were taken before and after injection of $\frac{1}{15}$ gr. of strychnine sulphate. In no cases was there any effect on the pulse-rate or blood-pressure, or the rate and amplitude of the respirations. In the cases of auricular fibrillation, no effect was produced on the irregularity.

As the evidence that a single dose of strychnine is not beneficial in heart failure does not prove that its prolonged use may not be so, Newburgh investigated the effect of large doses given for several days in chronic heart failure. The investigation was confined to 8 patients who did not recover compensation after rest in bed. Strychnine was given in doses varying from 0·03 to 0·1 gr., and, in some cases, toxic effects were produced. None of the patients was benefited, though 4 subsequently recovered compensation under digitalis.

Newburgh concludes that neither pharmacological nor clinical evidence justifies the use of strychnine in acute or chronic heart failure.—*American Journ. of Med. Sciences*, p. 696

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

THE INFLUENCE OF HEAT AND COLD UPON THE HUMAN BODY.*

By R. FORTESCUE FOX, M.D., M.R.C.P., F.R.Met.S.

WHETHER it be through the medium of air or water, heat is always passing to the body or being abstracted from it. A constant interchange is going on, but heat is not simply added or taken away. The warm-blooded animal *reacts* to the impression of heat, with definite changes in the tissues. And since living forms can exist only within narrow limits of temperature, it should not be thought surprising that changes of heat in the medium, within those limits, can be turned to account in the care of health and the cure of disease. It has been abundantly shown that the physiological processes, both of animals and plants, are sensitive to changes of temperature.

LIFE AND WATER. PATHOGENIC ORGANISMS.

Water is still the *sine qua non* of life. The greater number of the cells of the body still carry on their functions in a watery medium. Even in their limits of temperature, water and life are at one. The limits of life may be placed at the points where water loses its liquid form—where it freezes and boils. Within these limits every living species has its own much more restricted space, and its own point of *optimum* temperature. Most warm-blooded animals die if their temperature is raised 100 or 12°. Plants obey the same law. Many of the pathogenic organisms can grow only within a very narrow range of heat (see Fig. 1), and it is remarkable that their *optimum* temperature should correspond with that of the human blood. Considering their sensitiveness to temperature it is not surprising

* Second Chadwick Public Lecture delivered at the Royal Society of Medicine (abbreviated).

HEAT LIMITS FOR GROWTH OF PATHOGENIC ORGANISMS.

(= optimum temperature.)

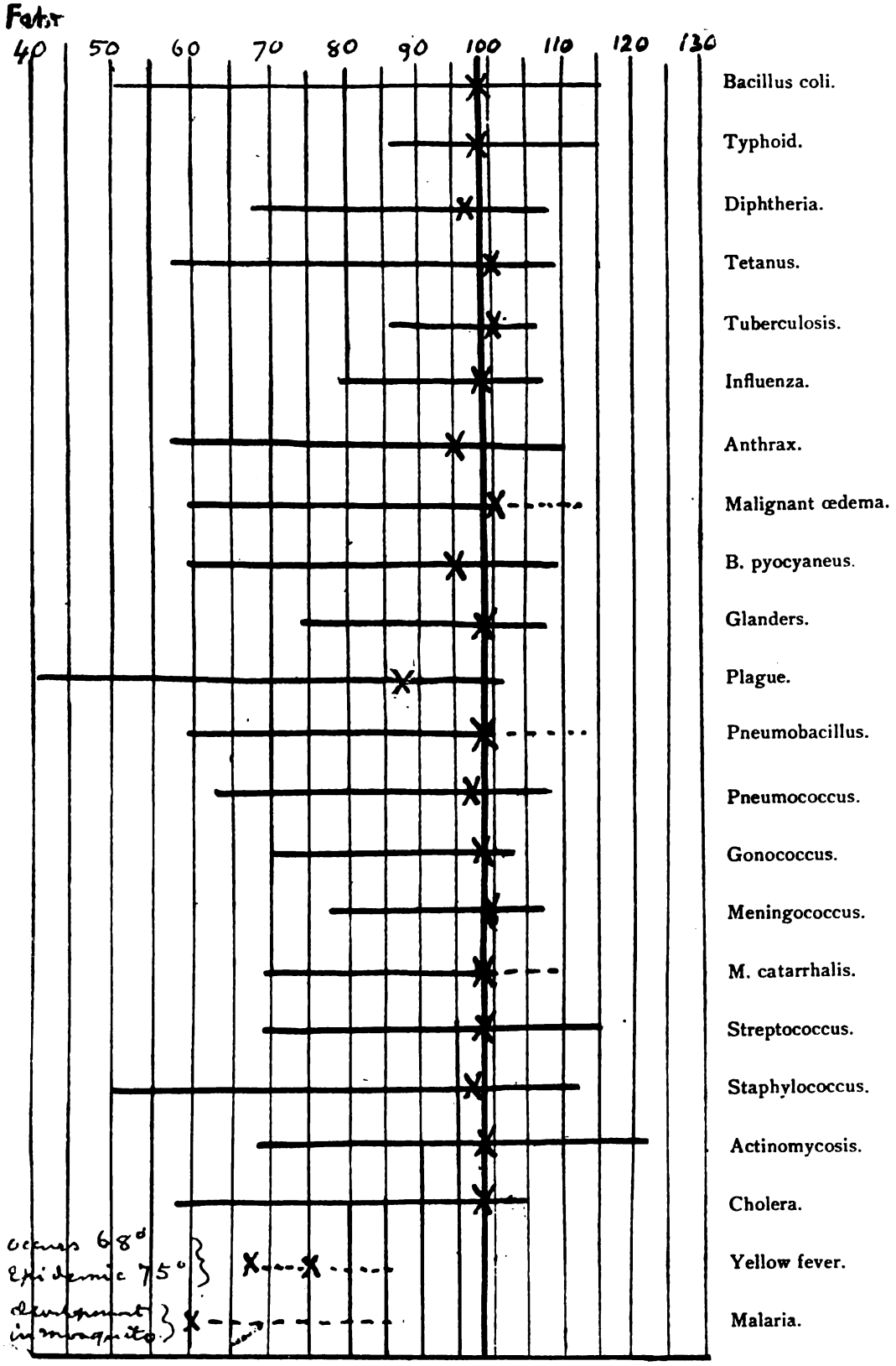

FIG. 1.

that microbes should be affected by climate and season, and even by changes of weather. Most pathogenic organisms take on a more intense activity in hot countries, and their virulence is no doubt affected by excess or deficiency of heat in the individual.

It follows from these facts that if the temperature of the human body could be temporarily raised a few degrees, the microbes of many parasitic diseases, if present in the body, would be destroyed, and this is no doubt the "intention" and the effect of pyrexial reactions.

Heat Production.

The healthy body must have an enormous power of heat generation, for even in London its temperature is on the average about 50° F. above that of the surrounding air; and in the arctic regions it may be maintained 190° F. higher than that of the atmosphere, for many months together. When well supplied with food man has an almost incredible power of withstanding cold. The normal daily heat production, which is sufficient to raise 55 lb. of water from freezing to boiling point, must be capable of increase to make good any possible loss.

Heat Loss and Regulation.

The loss of heat is minimised by the non-conducting cutaneous and subcutaneous barrier. The warmer the skin in relation to its surroundings, the greater will be the loss of heat. It follows that coolness of the surface is a protective and beneficial device, economical of the body heat, as in many persons whose heat production is deficient. In whales and other large animals in the arctic seas the surface of the body is very cold, but in man the skin temperature does not fall on the average more than 10° below that of the blood; and hence the necessity of protection by clothing. The warmth of the skin in man at any moment, and therefore the loss of bodily heat, is regulated by the opening or closing of the cutaneous circulation. So ample are the blood-vessels at the surface of the body that they could contain more than one half of the total quantity of blood. Both the blood-vessels and the sweat-glands are under nervous control—witness emotional states—and both are a necessary part of the heat-regulating mechanism of warm-blooded animals. The peripheral nervous system links up the skin reflexly with all other parts of the body. Consequently it has been said that impressions of heat and cold in baths act *directly* upon the skin, and *indirectly* upon all the organs and tissues. There is reason to believe that the unprotected skin of man is more sensitive to external impressions, and takes a more important part in physical and mental processes than that of any other animal.

The refreshing and reviving effect of cold is a familiar experience. The Highlander on the march finds that cool air about his knees prevents overheating and fatigue. In syncope or collapse the impression of cold sometimes restores the circulation and breathing by reflex action through the vaso-motor and respiratory centres. Thereby both consciousness and mental, as well as muscular power are revived. This can only signify that cold in its proper application is one of the most powerful stimulants known to science.

Modes and Media of Heat.

For man there are three possible kinds or *modes* of *heat*, according to their relation with the temperature of the skin. The average surface temperature, about 93° F., should be regarded as a great dividing line. Baths of all kinds below that line are charged with *negative* heat, above that line with *positive* heat, and at or about 93° F. the heat is *neutral* or "indifferent." The neutral point is not the same for all persons; it varies in disease, and its determination is an important matter in medical bathing.

Water is the best medium for subjecting the body to negative or positive heat, because it is a great heat carrier and a good conductor. Cool water has a veritable appetite for heat, and rapidly withdraws heat from bodies warmer than itself. The limits of application of heat in water are for negative heat 32° F., where water becomes a solid, and for positive heat about 120° F., where for most persons it becomes intolerably hot. *Moist air* and *vapour* are useful media for moderate degrees of heat, applied generally or locally, and in the Russian and Scandinavian vapour baths the upper limit rises to 135° F., or more, which is insufferably hot to unaccustomed persons. Much higher temperatures can be comfortably borne in *dry air*, as in the Turkish and other "hot-air baths."

Temporary Climates: the Reaction to Cold.

By such simple means as these it is an easy matter to subject the body, or any part of it, to a *change of climate*, at any desired degree of heat, negative or positive. Baths may be looked upon as artificial and temporary climates. Heat to one part of the body may be combined with cold to another, and one climate may be made to succeed to another. We have in baths a mode of treatment capable of eliciting from the body, or any part of it, the reaction or series of reactions belonging to thermal impressions.

Everyone may study for himself the *reaction of the body to cold.* Upon entering a cold bath there is an immediate contraction of the surface arterioles. The vascular network shrinks, with a sensation of "chill." The skin blanches. The blood, driven from the surface, is increased internally, especially in the abdomen. The breathing, after a momentary cessation, becomes deeper, and there is a great increase in the intake of oxygen and the output of carbonic acid gas. The pressure of the arterial blood rises, for which reason cold baths are often inadvisable in cases of increased blood-pressure. After these effects, which are proportional to the degree of cold, another series of effects are presently manifest. The centre for heat-production is stimulated and a great increase of oxidation takes place, especially in the muscles, liver, brain, and glands. In a cold bath the body is said to "shiver itself warm" by involuntary muscular contractions. Soon a sense of warmth replaces that of chill. The surface vessels relax and the blood comes swiftly back to the skin, which becomes pink in colour, if the phenomena of reaction are normal. The heart's action, at first quickened, becomes slower and stronger, and with the increase of heat and circulation there is an increase of vigour, both mentally and in the muscles.

The central fact in the reaction to cold is the generation of heat. The effect varies much with the nature of the bath and with attendant circumstances. Frictions, which usually follow the *morning bath*, greatly favour a healthy reaction. *Sea baths*, which are also often wave baths, sun baths, and wind baths, often show the effects of cold in an extreme degree, with faulty reaction. *Baths in flowing water*, like rivers, are also much more cooling than those in still water. Both with sea and river baths, short exposures, with subsequent friction and exercise, are generally requisite to ensure normal reaction. The table after Pariset (Fig. 2) shows the effect of baths at different temperatures, evidenced in the *reactions* of the nerve centres, the circulation, and the bodily heat.

It is difficult to believe how great an increase of combustion in the tissues may be induced in a

TABLE OF REACTIONS

FAHR.	BATHS	NERVE CENTRES	CIRCULATION	BODILY HEAT
120	VERY HOT (Hyper Thermal)	VERY STRONG	VERY STRONG	STRONG
105	HOT (Thermal)	STRONG	STRONG	MEDIUM
98	WARM (Sub Thermal)	MEDIUM	MEDIUM	FEEBLE
93	NEUTRAL (a-Thermal)	NIL	NIL	NIL
90	COOL	MEDIUM	MEDIUM	MEDIUM
75	COLD	STRONG	STRONG	STRONG
60	VERY COLD	VERY STRONG	VERY STRONG	VERY STRONG
32				

Fig. 2.

healthy person by short exposures to cold. Experiment has proved that a bath at 60° F. may in two and a half minutes more than double the intake of oxygen by the lungs, with an equivalent output of carbonic acid gas. The amount of heat generated in a cold bath may be increased twenty-four fold, and fat persons benefit from the combustion of fats. In this way the serious loss of heat in cold baths is at once made good; but unless the bath be very brief a great demand is made upon the reserves of energy.

Every kind of exposure to cold, by changes in wind or weather, by alteration of clothing or by baths, first of all checks the circulation at the surface of the body, sometimes with a subjective sensation of "chill." But the heat reserves of the body are at the same time called up, and a "glow" is experienced as the circulation returns to the skin, often with a sensation of energy and well being.

If it were not for this reaction, which is in continual operation, warm-blooded animals could not maintain their bodily heat. Cooling baths and applications only adapt to medical purposes a daily physiological process.

If the impression of cold is too great or too prolonged, the production of heat fails to keep pace with its loss. The temperature of the body begins to fall. The superficial veins become paralysed and dilated, which is indicated by a blue-red or mottled coloration of the skin. The body passes into algidity and collapse. Similar changes in the cutaneous circulation are seen locally in exposed portions of the body in wintry weather.

Whilst, therefore, up to a certain point cold is a powerful stimulant, it becomes a dangerous and fatal depressant in excess. In ordinary applications much depends on the power of reaction in the individual. A healthy reaction to cold can often be developed by training, especially in young persons. It is also powerfully promoted by a preliminary application of heat. That is why cold baths, and especially sea and other baths in the open air, are best taken when thoroughly heated (but not fatigued) by exercise; or, where that is impracticable, after a brief hot bath. The value of friction in promoting the return of the surface circulation has been already alluded to. The reaction is diminished and may be lost when the bather is already cool before cold is applied, when he is quiescent during and after the bath, and when the cold is excessive and prolonged. A cool bath without reaction is a physiological crime.*

Effects of Heat, Good and Bad.

The *reaction of the body to heat* is a familiar experience. The chilled body literally expands before an open fire, and hot applications may be the best of anodynes. It is recorded that nothing gave so great relief to the British troops after long exposure to cold, wet, and fatigue in the winter campaign in Flanders as the hot baths provided for them. Chill was removed, pain and stiffness removed, fatigue and exhaustion removed simply by heat. Heat proved a more powerful stimulant even than food. It is also recorded † that in the more serious condition of "fatigue fever," a toxæmia of excessive exertion, with extreme muscular pain and stiffness and often nervous symptoms, very hot baths have had the best results, relieving cramp, pain, and stiffness, restoring the use of the muscles and removing the more serious toxic symptoms.

Examples of the *beneficial effect* of positive heat, less or more in degree, local or general in application, could be indefinitely multiplied.

On the other hand, the *ill effects* of too much heat are equally familiar. Such are the depression, enervation, lassitude, disinclination for physical or mental effort, experienced in overheated rooms, in "relaxing" climates, and in long-continued sultry weather. It is true that individuals are very differently constituted in this respect. Some are never really well but in cool and "bracing" weather or climates, their vigour depending on the stimulus of cold. Others are unwell and shrunken in the winter, and only at their best in the heat.

The intolerance of cold by some persons and of heat by others often amounts to a serious inconvenience and is becoming more common. It is a disorder of the circulation and depends mainly upon habitual inactivity of the skin.

Reaction to Heat.

Heat and cold are not opposites in their effect upon the body. The cold bath of the Englishman may be contrasted with the hot bath of the

* See the writer's 'Principles and Practice of Medical Hydrology.'

† 'Brit. Med. Journ.,' 1915, vol. i, p. 206.

Japanese, but both are stimulants. The Japanese custom goes back for 1000 years, and probably the Japanese constitution has become inured in the course of generations to temperatures that are intolerable to Europeans. The daily bath is often taken at 120° F., and at Kusatsu the bathers enter the water at 123° F. or even hotter. In hot baths the superficial arterioles, after a momentary contraction, greatly expand. The skin becomes flushed and heated, from within as well as from without. The heart's action is accelerated, the blood-pressure reduced. A profuse perspiration is excited.

Winternitz and his pupils have shown that heat, as well as cold, increases the metabolism of the body. In a hot bath the intake of oxygen and output of carbonic acid gas may far exceed that which occurs in fever (111 per cent., as compared with 20 per cent. over normal). The fact that heat may actually *increase* the intrinsic heat production has a very important bearing on the use of baths. When the body is immersed in hot water the normal loss of heat cannot take place; the heat-regulating function is therefore thrown out of action, and the body temperature rises. The temperature of the skin soon exceeds that of the internal parts, which is entirely contrary to the natural condition. Meanwhile, the production of heat is increased and it accumulates more and more in the body. The first stimulating effect of heat gives place to feverish excitement. An artificial fever—a true bath fever—is in fact produced, and sometimes very rapidly, by the double process—increased formation and diminished loss. How quickly the bath fever is developed is shown by the fact that in a bath at 104° C. the temperature of the blood may rise as much as 5 degrees in half an hour. How long it may continue is indicated by the observation (of Wick) that the normal temperature of the skin is often not regained two hours after the bath, and that the consumption of oxygen was still 29 per cent. above normal one hour and a quarter after the bath (Winternitz).

Thermal Debility.

However beneficial brief hot baths and brief fevers may be, it must be remembered that no warm-blooded animal can live for more than a short time in a medium warmer than its own blood. The physiological facts as to the effects of heat upon the human metabolism throw much light on the action of hot baths in chronic disease and also go far to explain the ill effects that may follow upon their incautious use.

If the hot bath exceed a certain point of duration, a condition of enervation and debility follows, such as sometimes follows other forms of pyrexia. This may persist for many hours or even days. It is more or less proportional to the degree as well as duration of the heat. Those who persistently take hot baths, unless of very brief duration, commonly suffer from permanent loss of tone in heart and circulation (vaso-motor paresis) with enfeeblement of the nerve centres generally. The incautious practice of hot bathing may bring about a condition analogous to the thermal fever or heat stroke met with in hot, moist countries. In both cases the action of the skin is dangerously checked and heat accumulates in the body.

Cold after Heat.

The Japanese are the most scientific of bathers, for their hot baths are very brief. They enjoy the first and stimulating effects of heat and quit the bath before the later and injurious action has commenced. Happily, it is possible to obviate and forestall these ill-effects by the free use of cold after heat, as the Japanese are accustomed to do. Cold applications have for their immediate action to restore the tone of the circulation, to slow and strengthen the action of the heart, and to invigorate the nerve centres. Hence, "cold after heat" is the golden rule that ought to be inscribed in every bath-room. It is the only safe rule for hot baths, whether hygienic or medical, especially in the case of young and elderly persons and in those who suffer from cardio-vascular affections.

An admirable example of cold after heat is afforded by the vapour bath that has been used in parts of Russia and other northern countries since prehistoric times. Whole families repair to a small building filled with steam at a much higher temperature than could be tolerated by unaccustomed persons. The seats are sometimes arranged in tiers, the highest places being of course the hottest. The steam is produced by pouring water over hot stones or iron shot. When sweating has set in, the bathers are switched with birch twigs and afterwards rubbed with fats and soap-suds, a process interspersed with more vigorous frictions and

kneadings. Perhaps an hour is so spent by the Swede, Fin, Esquimaux, or Russian, and then he comes out with a red-hot skin bathed in perspiration, rolls in the snow or ice-cold water, and so runs back to his house. Such baths are esteemed by these people a sure protection against disease. The ordinary Turkish bath, as well as the ancient baths of Greece and Rome, are examples of the same salutary principle.

Vaso-motor Atony and Hypertonus.

Cold after heat is a practice well adapted for cold and temperate countries. Without the free vascular expansion induced by heat it is impossible to obtain the whole tonic effects of cold. The colder the climate the more necessary becomes the initial relaxation of heat; the greater also is the amplitude of the vaso-motor oscillation. The heart of the Greenlander is said to beat only 30 or 40 times to the minute. There is a condition of physiological *hypertonus*. How different is the condition of the circulation in the tropics! Persons unaccustomed to heat are already in a state of incipient fever. There is vaso-motor atony. The pulse is soft and rapid and the blood-pressure is reduced. Hence it is that persons with a feverish tendency are not well in the tropics or in hot weather.* It follows that, in general, vaso-motor atony should be treated by cold and hypertonus by heat. The first condition is characteristic of acute, the second of chronic disease.

Protection against Cold and Heat.

Comparing extreme heat with extreme cold in their relation to mankind, one great difference is evident. Man can protect himself against almost any degree of external cold by checking the loss of heat with artificial coverings and by the physiological increase of heat production. According to the degree of external cold, more and more heat is produced and liberated within the body. Explorers in the Arctic have said that when they enter upon a very cold region enormous quantities of food are served out, and that cold powerfully stimulates both appetite and digestion, especially for the "fuel foods."

On the other hand, man has but few and imperfect means for protecting himself against excessive heat. If the air is not too moist and is *moving freely*, the loss of heat by perspiration may be adequate. But if the atmosphere is stagnant, the fan or punkah becomes a life-saving device, and it is necessary to reduce the production of bodily heat to a minimum by an inactive life and low diet.

Neutral or A-thermal Baths.

The heat of very hot climates and of hot baths is in excess of that of the body-surface, and has, therefore, been distinguished by a *plus* or positive sign. On the other hand, the degree of heat in cold climates and in cooling baths is less than that of the body, and is distinguished by a *minus* or negative sign. But between the positive and the negative there is, both for climates and baths, a neutral point. At a certain temperature the functions of heat production and loss are unaffected. The body is in a state of thermal equilibrium.

The neutral point or point of *thermal indifference* varies with the medium. Some media convey and absorb heat better than others. The point of thermal indifference for air is said to be about 85° F. This is the temperature of the air included in ordinary clothing. Let the temperature rise, the loss and production of heat are diminished; if it falls they are increased, but at this particular point the normal exchange is undisturbed. In some tropical countries men live always in an atmosphere approaching this temperature and with a minimum amount of clothing. Their bodies are in a state of thermal equilibrium, and the delicate mechanism for the regulation of heat production and loss, upon which man depends for his existence in cold and variable climates, is more or less superfluous. The reactive power of the body to cold is seldom exercised and imperfectly developed. Hence the risk of cold applications in hot countries.

Water conducts and absorbs heat much more freely than air. In a bath at about the temperature of the skin (93° F.) heat production and loss are unaffected. The *point of thermal indifference for water* is, therefore, fixed at 93° F. Hotter or colder baths (+ or −) always affect the bodily heat; these, not at all. It follows that when, for purposes of cleanliness or otherwise, it is desirable that baths should be prolonged or frequently repeated, they ought to be at or near the point of thermal indifference (93° F.), otherwise they

* See Huggard, 'Handbook of Climatic Treatment,' 1906, p. 80.

will add to or diminish the bodily heat in an undesired and possibly injurious manner.

A good example of the thermally-indifferent or a-thermal bath is the natural bath of Kawanaka, near Ikao, in Japan. The temperature is a few degrees below blood heat, and the bathers at Kawanaka can remain in the water for a month, having stones across their knees to prevent them floating in sleep. It was said that a caretaker, a hale old man of 70 years, remained in the bath the entire winter. Similarly, in certain European waters, such as Leukerbad, the bathers remain immersed for several hours, and refreshments are served on floating tables.

Effects of A-thermal and Sub-thermal Baths.

In baths of this kind the body exchanges a gaseous atmosphere for a watery one, and, since all life was originally aquatic, the a-thermal bath may be looked upon as a reversion to type. Moreover, it substitutes one massive equable nervous impression for a succession of many changing impressions on the sensitive body surface. It is, therefore, always sedative. A difference of one degree in the temperature of the bath may materially alter its action. What has been called the "Nauheim Bath" is a bath of this description, in which the action is modified by the presence of salts and gases.

Baths within a very narrow range of heat, above or below the average temperature of the skin, may be *a-thermal* or *sub-thermal*; that is to say, the thermal action is altogether absent or slight and gradual. From inequalities in the surface temperature the action may be present in one part of the body and absent from another. Clinically these more or less neutral baths are of great medical value and wide application, in irritability and atonic conditions of the nerve centres, heart, and circulation, in advanced life, and in convalescence.

The Differentia of Waters.

The effect of temperature lies at the foundation of all bath treatments. Heat must always be the most important factor in every description of bath. All waters have a certain character in common. But in addition to the qualities in which they agree, how important and interesting are those in which they differ! The natural history of waters reads almost like a fairy tale. Sometimes they come up hot from the earth, charged with certain groups of elements—metals, metalloids, and gases—and having also physical properties, like radio-activity, not at present fully understood. Sometimes they carry in a minute state of subdivision, and in a colloidal condition, which baffles ordinary analysis, particles that have been proved to exert powerful antiseptic effects. Some have elements that are not otherwise employed in medicine. Some are so saturated with salts that they are only applicable for surface treatment; others have these salts in proportions that make them stimulating or soothing to mucous membranes. Other waters, again, are of a density or "tonicity" resembling that of the blood, and have been, therefore, described as "natural sera." The chemists have endeavoured to imitate the natural fluids of the body with artificial "neutral saline solutions," because "isotonic waters" have an affinity with the tissues. By the same reasoning, pure or distilled water, which is unknown in Nature, is anathema to the human body. And, lastly, other waters have an action which cannot be reproduced, owing to a particular combination of their constituents.

Enough, perhaps, has been said to indicate that by means of waters—in their internal and external application—a wide range of actions and reactions can be induced in the body, and that their scientific use constitutes an indispensable branch of medical treatment.

November 1st, 1915.

Appendicular Abscess in an Infant.—Phélip records a case in a female infant, aged 13 months, bottle-fed, who had suffered from constipation for two months. The symptoms were fever, diarrhœa, blood in the stools, and distension of the abdomen. A swelling could be felt in the right iliac fossa per abdomen and per rectum. The diagnosis lay between appendicitis and intussusception. A lateral laparotomy under chloroform was performed, and an appendicular abscess was found with a free stercoral calculus. Recovery was complete.—*Brit. Journ. Child. Dis.*, p. 313.

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We regret that owing to the war the number of pages must be diminished for a time.

PSEUDO-COXALGIA.

By E. MUIRHEAD LITTLE, F.R.C.S.,

Surgeon to the Royal National Orthopædic Hospital, London.

NOT many years ago any chronic affection of the hip-joint in childhood accompanied by limping and marked limitation of movement was likely to be put down to tuberculosis and treated as such with prolonged rest, etc. Such cases as followed a benign course and recovered with very little shortening or diminution of the range of movement, were naturally considered as instances of successful early treatment.

The X rays have been in use as helps to diagnosis for the last 20 years, but it is only during the last 10 years that their extensive use has taught us that some at least of the cases above referred to present features unlike those of tuberculous disease, and follow a definite course which is fairly characteristic.

In 1910 Calvé described a disease which he called pseudo-coxalgia. Perthes later recognised that some cases which he had classified as arthritis deformans juvenilis were really examples of another affection to which he gave the portentous name of osteo-chondritis deformans juvenilis.

There does not appear to be any clear dividing line between the conditions described by these two authors and the writer prefers to use the name "pseudo-coxalgia" because the pathology of the disease is obscure and this title does not commit us to any ætiology.

A typical history is that a child, between the ages of 3 and 12 years, apparently in perfect health, begins to limp and perhaps complains of some pain which, however, is never severe. These symptoms are not very marked, but on examination more characteristic ones are found. Flexion and extension of the thigh on the pelvis are free and

painless, but the range of the movement of rotation especially inwards is much below the normal, and abduction is well-nigh impossible, except under an anæsthetic.

In short: the movements of the joint which in health are those of a ball and socket are found to be reduced to those of a simple hinge-joint such as the elbow.

The lower limbs are equal in length at first, but later there may be some shortening. There is little or no tenderness on palpation of the region of the hip-joint and no tumefaction, but generally it is noted that the great trochanter on the affected side is unduly prominent. This appearance is probable due to slight wasting of the muscles on the affected side, which is a common symptom. The Trendelenberg sign is usually found. This consists in the drooping of the *sound* side of the pelvis when the patient stands on the foot of the affected side, holding the foot of the sound limb off the ground. In a normal child the buttock will be seen not to drop, but in congenital dislocation of the hip and some other conditions the abductors of the thigh are unable to hold up the pelvis.

Von Pirquet's or other tests for tuberculosis give negative results, unless, of course, there is or has been some tuberculous focus in another part of the body.

The most striking characteristic of the disease is revealed only by the X rays—for in this country, at any rate, no one is likely to emulate a German surgeon, who excised the head of the femur and thus secured an interesting and instructive specimen at his patient's expense.

In a typical case the upper border of the bony nucleus of the femoral head, instead of being represented by a segment of a circle, is shown as a flat surface (see Fig. 4). In photographic prints from the plate light spots are to be seen in the head, representing parts more pervious to the rays than the rest of it, or the bony nucleus may even be divided into two or more portions. The neck appears to be more pervious to the rays than its fellow, and this permeability is irregular, so as almost to suggest the presence of cysts. The epiphyseal line between head and neck is irregular and widened, and the outline of the acetabulum is blurred or irregular. Probably these light spots are due to islets of cartilage, such as were found in the only specimen of which an account has been published. The process is a chronic one, lasting for 2 or 3 years or even more, at the end of which time the patient has lost his limp, but he may be left with some shortening, and some reduction of the range of abduction and of rotation.

The disease is not very common. Delitala, who has collected records of 50 published cases, found only 6 cases out of "1500 cases of hip affection of orthopædic nature" which had been treated at the Rizzoli Institute at Bologna.

Of the 50 known cases, 40 were boys and 10 girls.

The affection is almost always unilateral.

Nothing certain is known of the causes of the malady. It has been suggested that hypothyroidism is to blame, and in one of the present writer's cases there was a family history of this defect.

As to treatment: The point of most importance is to recognise and not to mistake it for tuberculosis. This mistake may easily be avoided if the X rays are used. The characteristic flattening is unlike anything to be found in the early stages of morbus coxæ, and in the latter disease marked bone changes are accompanied by very pronounced symptoms, including loss of movement in all directions, and especially in the direction of flexion and extension.

Most surgeons who have dealt with this malady advise that no treatment should be adopted, and that the patient should be allowed to go about as usual without any mechanical appliance. It appears to the writer, however, that it is at least desirable to attempt to abduct the thigh and keep it in marked abduction, so that the head of the femur may not take a form as growth proceeds which would prevent this movement, when the active disorder is at an end. For this reason it seems advisable as soon as the nature of the trouble is ascertained, to abduct the thigh under anæsthesia and to fix it in that position by means of plaster of Paris or an abduction splint. With a patten or high boot on the other foot, the child is to be allowed and encouraged to move about freely. Fresh air and sunshine are no doubt desirable as they are for all children, but no drugs are indicated, unless it be thought worth while to try the effect of thyroid feeding.

Besides tuberculosis there are other diseases of the hip in childhood from which it is necessary to distinguish pseudo-coxalgia.

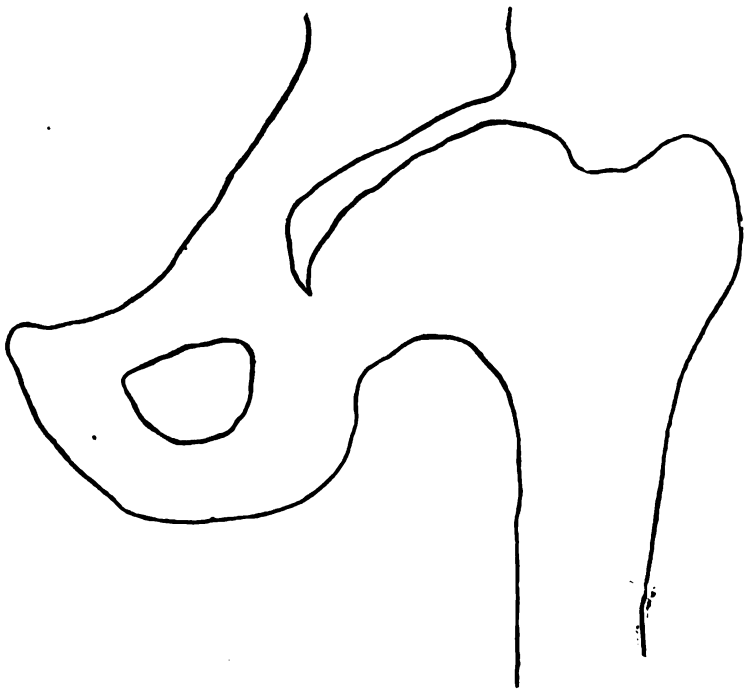

FIG. 1.—Outline of X-ray plate of L. S—, characteristic of unilateral coxa vara adolescentium, showing bending and deformity of neck and head of femur.

FIG. 2.—Outline of X-ray plate from an unmarried woman æt. 35 years, in whom both hips had been affected with coxa vara. She could not separate her knees more than ten inches, but suffered no inconvenience. She consulted me as to the advisability of matrimony.

These are coxa vara (see Figs. 1 and 2), cystic disease of the neck of the femur (see Fig. 3), and congenital dislocation of the hip. It is not necessary to go into the symptoms of these diseases, because with the X rays the distinction can be made with certainty in any case, and quite apart from symptoms. Acute and chronic rheumatic affections are not likely to be mistaken for pseudo-coxalgia, but there is one form of arthritis, of which more is heard in Germany than in this country, with which Perthes's earlier cases were confused. This is the disease called arthritis deformans juvenilis. In this disease the pain, the loss of movement in all directions, and the resulting deformity as well as the X-ray picture should prevent any mistake being made.

The following are the notes of two cases of pseudo-coxalgia which have come under my care:

CASE 1.—Male, æt. 9 years, a patient of Dr. J. Easton Scott, of Wallington, who has kindly furnished the following history: "Has quite a good health record, except for minor ailments. The immediate family history is good, but there seems to be a considerable element of hypothyroidism in the family. He was first seen by me in November, 1913, when I was told that he had been limping for 3 or 4 weeks. He complained of no pain in the hip or knee, but on manipulating the limb, he said it hurt him a little

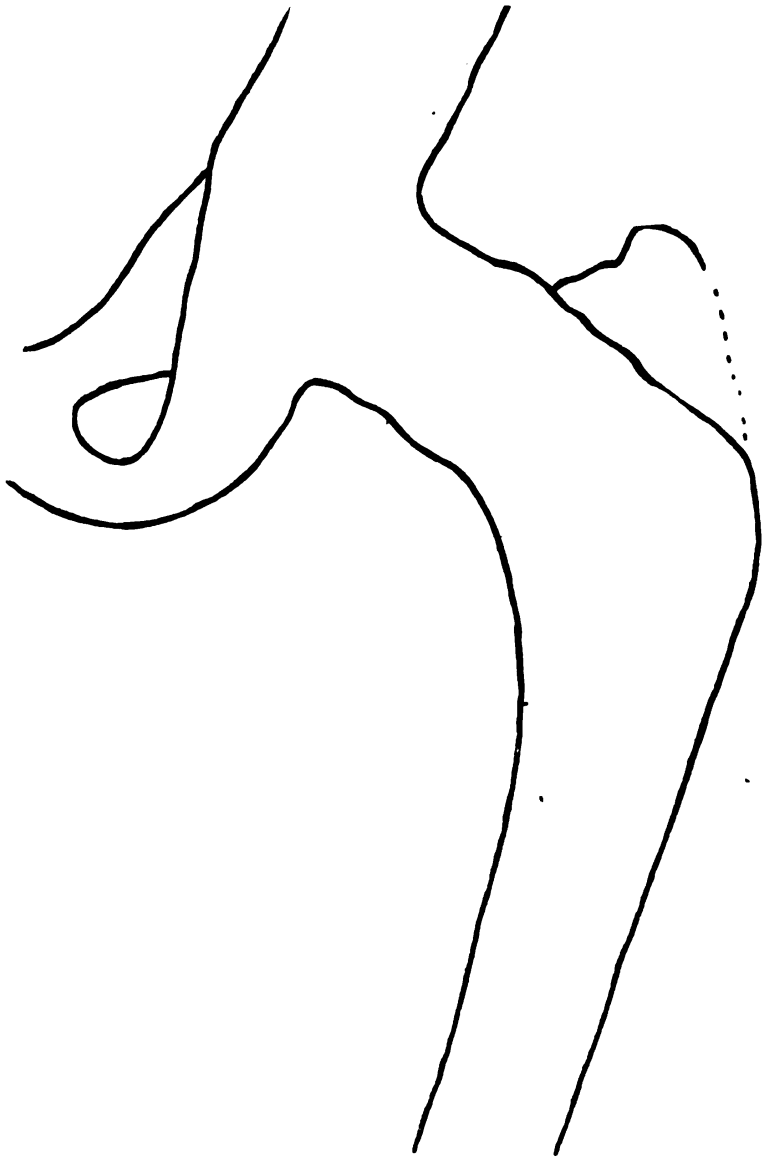

FIG. 3.—Outline of X-ray plate of unilateral cystic osteitis of the neck of the femur with softening and deformity. Reduction of the proportion of lime salts in the bones made X-ray pictures faint and details difficult to follow through tracing paper.

on the inner side of the thigh. No shortening, no wasting, and no alteration in the gluteal folds. There was, however, decided restriction of rotatory movement in the hip-joint. Flexion and extension were not impaired. I kept him about 6 weeks in bed, and the restriction in the rotatory movement seemed to disappear. I let him up, and, though there was still no pain and one could jar the limb and even get him to jump with it without any pain whatever, the limp was as bad as ever in a couple of days. He could, however, walk without a limp as long as his mind was kept on what he was doing."

When first seen by the present writer in April, 1914, the right lower limb was found to be a little longer than its fellow. All movements were painless, but abduction and rotation were not possible to the normal extent, and were resisted. Each trochanter was just below Nelaton's line. There was no symptom of sacro-iliac disease. The boy could not straddle properly. When he attempted to do so there was no abduction of the right thigh,

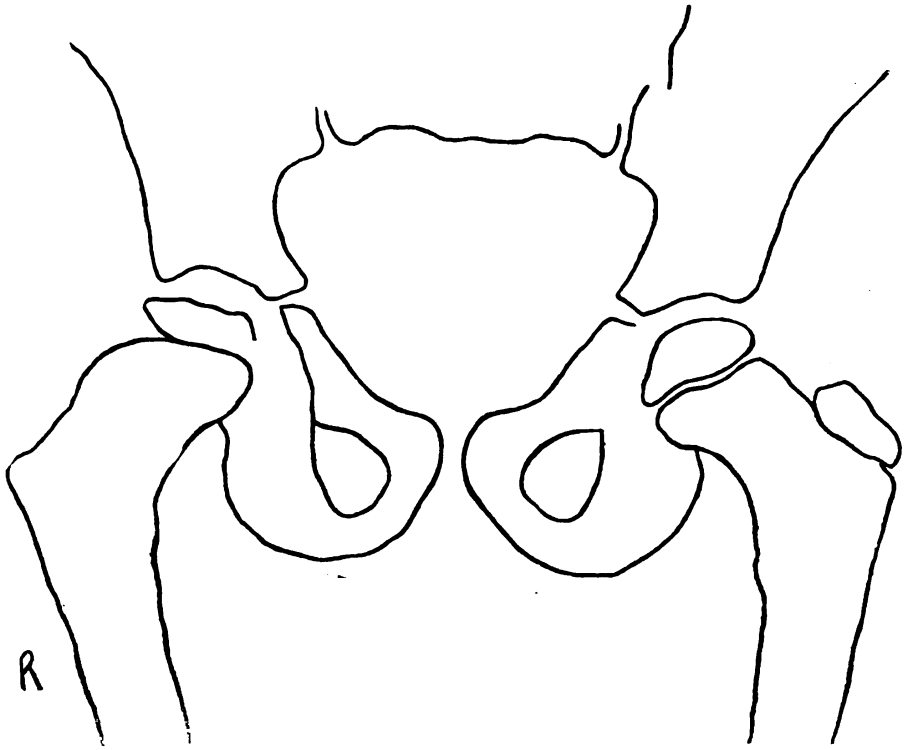

FIG. 4.—Pseudo-coxalgia. Outline of X-ray plate taken in April, 1914, showing characteristic flattening of the bony head of the femur.

but the trunk was held over the right limb. An X-ray plate (see Fig. 4) showed marked flattening of the head of the right femur and some indistinctness of outline of the right acetabulum. No pain was complained of on examination.

On April 21st, 1914, an anæsthetic was administered, and it was then found that abduction, nearly to the normal extent, was possible. A short gypsum spica was applied in the abducted position. The boy was ordered to wear a high boot on the left foot and to get about, and thyroid feeding was prescribed.

In July, 1914, a new X-ray picture showed more deformity of the head of the femur and more blurring of the acetabulum. The limb, however, could be passively abducted as far as 102 degrees, which is 12 degrees beyond the mid-position (*i.e.* the position in which the axis of the limb forms a right angle with a line joining the two anterior superior iliac spines). Full flexion was not possible. Tenderness was complained of upon pressure over the head of the right femur in the groin, and pain was referred to the same spot when the limb was forcibly hyperextended. No rotation was possible. The limbs were equal in length. A Thomas's thigh splint in the abducted position and a patten and crutches were ordered.

In February, 1915, abduction and flexion were nearly normal in extent, but very little rotation was possible.

An X-ray plate (see Fig. 5) showed that the head of the femur was still rather flattened and irregular in outline, although it was more rounded than it was in the first plate. There are still light spots in the print, suggesting the presence of islets in the epiphysis.

In June, 1915, all movements were good except rotation, which, however, was improved. The boy is growing fast. He runs about actively. The X-ray appearances are much the same as in February, but the bone seems to be more dense. There is no evidence of coxa vara. As measured on the X-ray print the angle of the neck on the right side is slightly greater than on the left. The right limb is not more than $\frac{1}{8}$ in. shorter than the left.

CASE 2.—Male, æt. 5 years, seen at the Orthopædic Hospital in October, 1914. This patient was sent to me by Dr. G. W. R. Skeene, of Brondesbury Park.

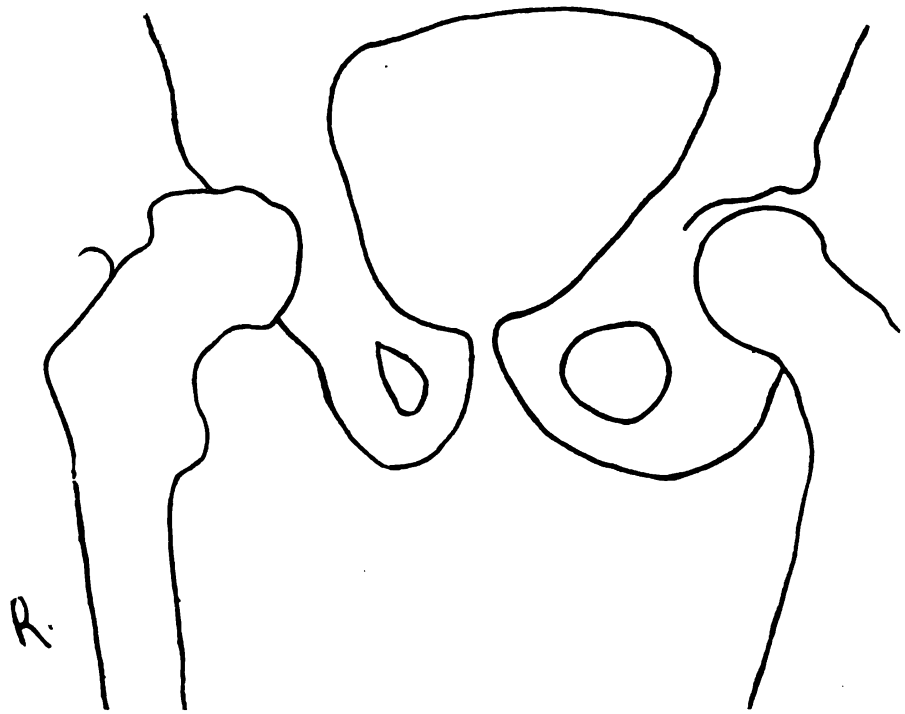

FIG. 5.—Pseudo-coxalgia from the same case as Fig. 4, but taken in February, 1915, showing improvement in shape of epiphysis.

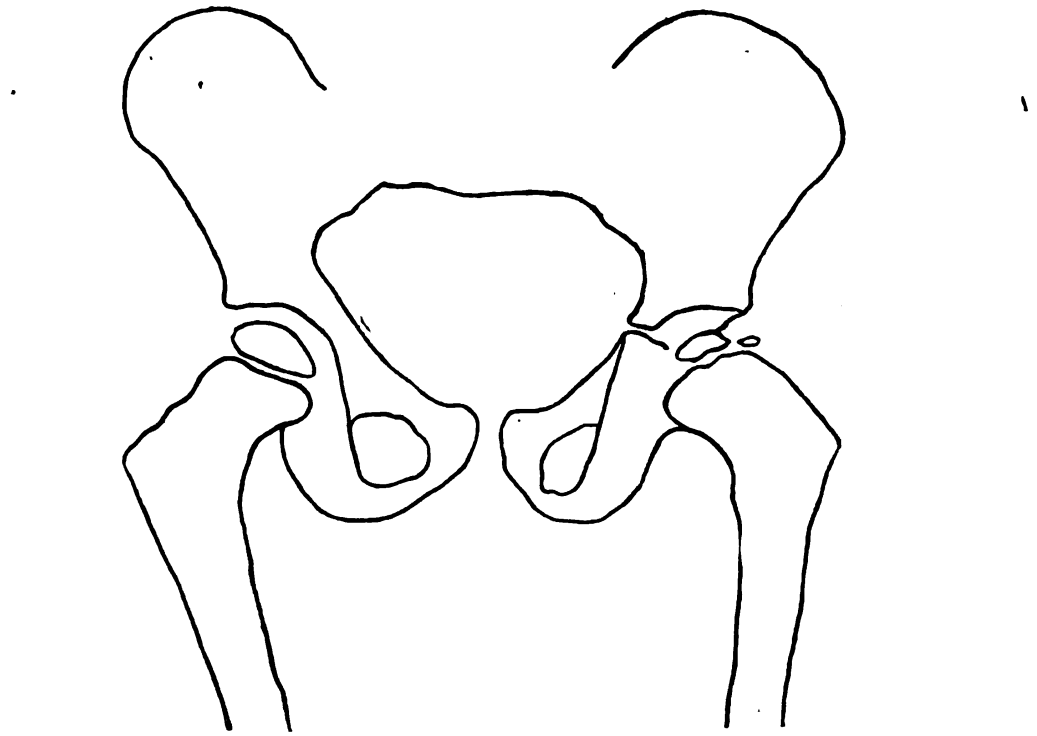

FIG. 6.—Pseudo-coxalgia. Outline of X-ray plate shows marked deformity, but not so characteristic as in Case 1.

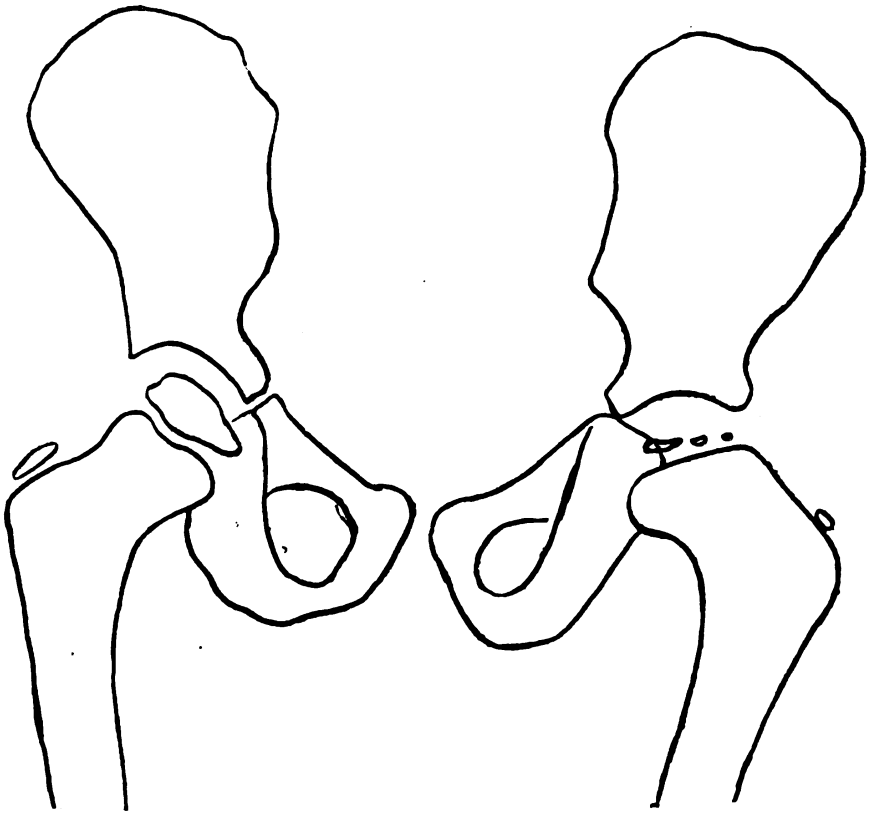

FIG. 7.—The same case as Fig. 6, three months later. Shows bony nucleus of epiphysis of left femur much smaller and broken up into three portions. Note also the small size of the nucleus of the great trochanter. The bony nucleus of the right femur is abnormal also.

History.—Occasional lameness has been complained of for 4 months or so. Occasional pain has been referred to the front of the left hip-joint.

On examination the limbs are equal in length, the left gluteal fold is much less marked than the right. Limited rotation and abduction of the left thigh. Complaint of pain on attempts to abduct fully. He runs actively without complaint. There is a slight droop of the left side in walking.

X rays showed (see Fig. 6) defective development of the head of the left femur, the epiphysis of which is much smaller and flatter than on the right side, and it is irregular in form towards its outer border. He was ordered to practise abduction and straddling.

On November 13th, 1914, the limb was ¼ in. shorter than its fellow. To relieve the femur of weight a Thomas's knee splint for the left limb and a patten for the right were ordered.

In January, 1915, he was walking well. There was no pain at any time. An X-ray plate taken at this time shows that there was very little bony epiphysis visible, and the pelvic wall of the acetabulum appeared very thin (Fig. 7).

In June, 1915, flexion and extension were free. A little rotation was possible. Abduction 5 degrees and adduction 15 degrees—a range of movement of 20 degrees.

On July 1st, 1915, X rays showed the appearance seen in Fig. 7. The bony nucleus of the head of the femur was represented by two or three small spots. Clinically, however, the boy had improved. The range of ab- and ad-duction was increased to 35°. There was no pain or disability despite the muscular wasting. All instruments were ordered to be abandoned. He ran about actively and seemed well.

Note.—Although this case did not show quite typical skiagraphic appearances the epiphysis was thinned, and the clinical symptoms were typical of pseudo-coxalgia. It is probable that the cartilaginous head is much less deformed than its bony nucleus.

November 8th, 1915.

SCLERO-CORNEAL TREPHINING IN THE OPERATIVE TREATMENT OF GLAUCOMA. By ROBERT H. ELLIOT, I.M.S. Second Ed. London : George Pulman & Sons, Ltd., 1914.

The first edition of this most valuable and instructive monograph appeared in 1913, and before it had been eight months in print the publishers were asking for a second edition, which appeared in the following year. The author has incorporated in this edition a digest of the recent additions to our knowledge of glaucoma, with the consequence that he has had practically to re-write the book.

Although Colonel Elliot is not the originator of 'Trephining the Sclerotic for Glaucoma,' he can claim to be the first to place the subject on a bedrock on which future workers may safely build. He has spared no pains in bringing the treatment of this disease up to date. An important feature of the book is that the work of others has been drawn on freely, so that the monograph is not one-sided, as so frequently obtains in similar treatises. Minute details are given of the technique which the author has, after very extensive and unique experience, found most suitable in carrying this valuable method of treatment to a successful issue. We heartily recommend Elliot's 'Sclero-Corneal Trephining' to everyone who has to deal with the dreadful malady in question. We feel sure that a careful perusal will lead to the adoption, as a rule, of this operation in preference to simple iridectomy. Speaking for ouselves, our first information of the technique of the operation was derived from the first edition, and, although we have since then wandered away from the details of the author's technique, the operation we now perform is essentially that described by Elliot.

The book is a credit to British Ophthalmology, and is another of the many examples in which the British surgeon can show his superiority over the Teuton in devising and perfecting new scientific methods. No up-to-date ophthalmic surgeon can afford to be without this valuable work. We recommend it as a reliable guide to an intricate problem of therapeutics.

PRACTICAL PRESCRIBING AND TREATMENT IN THE DISEASES OF INFANTS AND CHILDREN. By D. M. MACDONALD, M.D., F.R.C.P.E. Pp. 200. London: Oxford Medical Publications, Hodder & Stoughton, 1915. Price 5*s.* net.

This book is a companion volume to the 'Practitioner's Practical Prescriber' published last year and devoted especially to infants and children. It contains a posological table for children—a most useful item—and chapters on "Points in Prescribing," "Therapeutic Index," "Emergencies," "Points in Practice," as well as "Notes on Drugs" and the new B.P. It thus covers the whole ground in general practice. The value of the book is enhanced by a chapter on "Eye Diseases in Children" by Dr. Macgillivray, of Dundee, also one on "Tuberculosis in General Practice" by Dr. Halliday Sutherland, Consulting Tuberculosis Officer for North Marylebone and Medical Officer to the St. Marylebone Tuberculosis Dispensary, London, which will repay reading by the general practitioner. We can heartily recommend the book.

TOWARDS RACIAL HEALTH : A HANDBOOK FOR PARENTS, TEACHERS, AND SOCIAL WORKERS ON THE TRAINING OF BOYS AND GIRLS. By NORAH H. MARCH, B.Sc. Pp. 246. London : George Routledge & Sons, 1915. Price 3*s.* 6*d.* net.

To write a book on this subject which shall be successful in the best sense of the word is no easy task, and the best compliment to be paid to the author is that she has fully accomplished her task. There is a foreword by Professor Arthur Thomson, of Aberdeen University, which fitly appraises the distinctive features of the book, and which heartily recommends it as a wise and sympathetic contribution towards breaking down the barrier of the conspiracy of silence. The pages are entirely free from false modesty and cant, and the parts which merit it, treated with delicacy, without prudishness, almost with reverence. The book may safely be recommended to whom are committed the responsibility for the education of the young.

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IRREGULARITY OF THE HEART-BEAT.*

By C. H. CATTLE, M.D., F.R.C.P.Lond.,
Physician to the Nottingham General Hospital.

THE expression "irregularity of the pulse" is not synonymous with "irregularity of the heart." The radial pulse may beat with perfect regularity, yet on auscultating the heart certain arhythmical beats may often be heard, but which fail to transmit a pulse-wave to the wrist. The varying time intervals between the beats which indicate the arhythmia may affect the whole heart, auricles and ventricles, or there may be dissociated action between the two sets of chambers. The sounds of the heart which are so familiar to us are evidence of the mechanism of the ventricles only, but by the use of Mackenzie's polygraph, by which tracings of the venous pulse may be obtained, we obtain evidence as to what is taking place in the auricles. Taking the word irregularity in its widest sense we thus see that disturbances of rhythm may occur which are evident neither to palpation nor auscultation.

(1) *Irregularity due to premature systole.*—This form of disturbance often occurs in hearts which are for all practical purposes sound. It may occur in diseased hearts, but in itself, it is no evidence of disease. The common form of intermittent pulse, frequently attributed to indigestion, flatulence or neurosis is of this type. The "intermission" is due to two heart-beats occurring in rapid succession, the second or "premature" beat failing to affect the radial pulse. The second beat—constituting the premature contraction—may involve both auricle and ventricle, or more commonly the ventricle only. On auscultating the heart in which premature systoles occur it will be found that abortive heart-

* The substance of this paper was given with lantern demonstration before the Nottingham Medico-Chirurgical Society.

sounds can be heard corresponding in time with the intermission of the pulse. The sounds are weaker than the normal heart-sounds, and sometimes the premature contraction only produces one sound, the second sound being absent, owing to the feeble contraction failing to raise the semilunar valves. The premature contraction is followed by a compensatory pause of such a length that the time occupied by the normal and the premature contraction is exactly equal to the period occupied by two normal beats. The ventricle having enjoyed an extra long rest, the next cardiac contraction following the disturbance is more forcible than normal. Frequently the coupled heart-beats and the compensatory pause can be felt on palpating the region of the heart's apex. These points are illustrated in the following tracing taken from Dr. Mackenzie's 'Diseases of the Heart' (Fig. 1).

Premature systoles rarely occur below the age of 20, but are common after middle life. In many cases there is no obvious change in the cardiac muscle or nerves, but there is no doubt the affection is in some way connected with advancing years. Nevertheless, many elderly people are capable of considerable exertion, in spite of a tendency to premature systole. I know the case of a gentleman who for many years has been subject to premature systoles. Although now in his 61st year he can cycle 50 miles a day when on his holiday, and keep it up day after day for several weeks. An irregularity due to simple premature systole can generally be distinguished from that accompanying organic heart disease. In the former case the pulse becomes regular with exercise, and in the latter exertion not only makes the pulse more irregular, but causes breathlessness and cardiac distress. Premature systoles generally reappear when the heart slows after exercise, and the irregular pulse of organic disease improves with rest. A sound digestion, freedom from worry and plenty of outdoor exercise are the best preventives of premature systole; while dyspepsia, constipation, nervous disturbance and town life are apt to give rise to the trouble. The patient's symptoms are often decidedly unpleasant. He may complain of fluttering, "stopping of the heart," and a sinking feeling. The more alarmed he is the more the disturbance increases, showing the effect of nervousness. These symptoms have often been mistaken for impending heart failure both by patients and doctors. On the other hand, some patients are subject to premature systole for years, and are unconscious of the fact. When consulted for premature systole we must endeavour to make quite sure the patient has no organic disease. If the heart's action

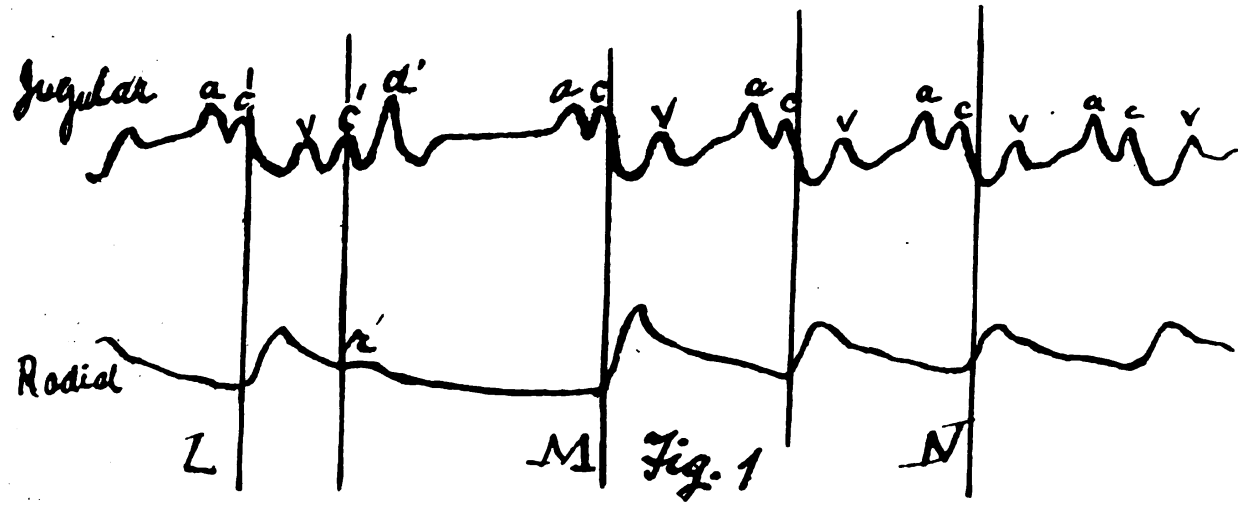

Fig. 1.—The upper tracing is taken from the jugular vein, simultaneously with the lower, taken from the radial. The letter *a* indicates the wave produced by the auricular contraction; *c* the carotid pulse, which just precedes the radial. The wave *c'* is caused by the premature systole in the carotid, and *r'* by the same in the radial: *a'* represents a normal auricular systole, not followed by ventricular systole. The period *L.M.* includes the intermission of the radial pulse, when the faint premature beat may or may not be perceptible. The period *L.M.* is of the same length as two normal beats, shown at *M.N.*

becomes regular and accelerated by making the patient take a brisk walk we may feel pretty sure his heart is intrinsically sound. Abnormalities of rate, of rhythm, of force, or the presence of murmurs are not in themselves evidence of heart failure. The real test of soundness or unsoundness of the heart, as Dr. James Mackenzie more than any other clinician has taught us, is the response the organ is capable of making when called on for increased effort. Patients suffering from premature systole, who show no evidence of cardiac failure, should not be told the heart is "weak" or be alarmed in any way. They will get well all the sooner if the medical attendant reassures them and advises them to notice the disturbance as little as possible.

But little drug treatment is required. The bromides are useful for their calmative influence over the nervous system. Small doses of belladonna sometimes cause the rhythm to become regular. If troublesome attacks of palpitation occur sal-volatile is useful.

If normal beats and premature systoles alternate the result is frequently a bigeminal pulse, *i. e.*, two pulse-beats in rapid succession and then a pause; or trigeminal, where the normal beat is followed by two premature ones and then a pause. Premature systole has relations with other forms of disordered mechanism, notably with paroxysmal tachycardia, though of course the latter disorder is rare compared with the former. Usually after the occurrence of one or two premature systoles the normal rhythm asserts itself. It is not uncommon for bigeminal or trigeminal rhythms to last for five or more minutes at one spell, and to recur at irregular intervals over a whole day. Without apparent cause the number of daily attacks may vary greatly. An attack of tachycardia, lasting hours or days, is nothing more than a long series of premature systoles. In patients liable to this disorder premature systoles can often be induced by telling them to breathe deeply or hold the breath.

(2) *Juvenile form of irregularity.*—In children and young adults the pulse can often be found to slow during expiration, and becomes faster during inspiration. In many adults the same phenomenon can be observed when they *voluntarily* breathe slowly and deeply; but in children the occurrence is involuntary. The condition is illustrated in the following figure (Fig. 2).

This alteration of rhythm is an entirely physiological phenomenon, depending on varying degrees of vagus influence. The vagus nerve, which slows and controls the action of the heart, appears to be stimulated during the act of expiration. The contraction of the heart commences in a nodule of vestigial tissue near the opening of the superior vena cava, and is for the most part automatic and idio-muscular; at the same time the influence of the vagus on the spot just mentioned, according as it is increased or decreased, is able to slow or quicken the action. The respiratory irregularity of the pulse is abolished by exercise and sometimes by holding the breath. This form of irregularity is, however, sometimes seen in young subjects quite apart from the influence of respiration; by means of the polygraph it can be distinguished from other forms of irregularity. Without the aid of this instrument irregularity occurring in childhood or youth, apart from obvious heart disease, may generally be regarded as belonging to this type. Respiratory irregularity is particularly apt to occur in children after febrile attacks. After diphtheria it might be mistaken for serious heart-weakness unless we are thoroughly alive to its nature, and it has sometimes been considered an evidence of perverted innervation in suspected

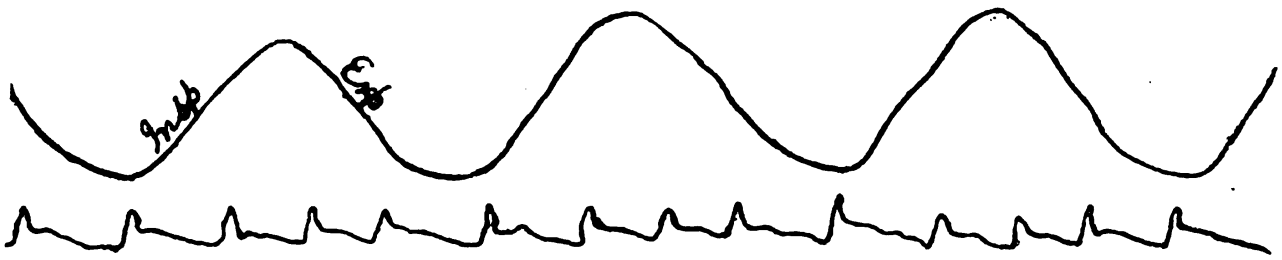

FIG. 2.—Youthful type of irregularity, showing quickening of the pulse with inspiration, and slowing with expiration. (Mackenzie.)

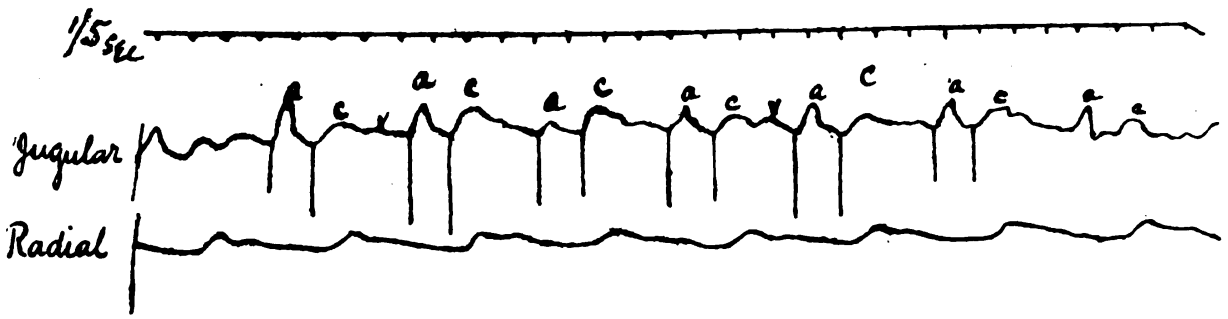

FIG. 3.—Partial heart-block due to digitalis. The *a c* interval is lengthened, but the tracing is otherwise normal. The patient had a mitral systolic bruit and dilated leftventricle.

meningitis. Dr. Mackenzie has described this form of irregularity as among the "manifestations of a healthy heart." Vagus influence is partially suspended during pyrexia; during convalescence this influence is temporarily exaggerated and causes the periodic slowing of the pulse. This phenomenon should always be looked for after any of the specific diseases of childhood. I have several times seen it well marked in young children after diphtheria, and have known it regarded by practitioners as a sign of heart-weakness. But if the case has reached a condition of convalescence in other respects this sign alone does not call for a period of prolonged rest.

(3) *Irregularity due to heart-block.*—The two preceding forms of irregularity commonly occur apart from organic heart disease, and are frequently met with in hearts which are for all practical purposes sound. Heart-block, on the other hand, is evidence of degeneration or functional impairment of a particular region of the cardiac musculature. By "heart-block" we understand a partial or complete blocking of impulses passing from the auricles to the ventricles. The contraction of the auricles is transmitted to the ventricles by the now well-known "bundle of His"—a strand of primitive fibres commencing in a nodule of tissue situated in the right auricle near the opening of the coronary vein. The destruction or functional impairment of this connecting band leads to dissociated action between auricles and ventricles. The normal relation between the contraction of the auricles and ventricles is represented in Fig. 1 by the letters *a c*. In a normal polygraphic tracing the period *a c* occupies $\frac{1}{5}$th second. When conductivity is impaired, the interval *a c* is lengthened; when it is arrested by destruction of the bundle, the ventricles fail to contract and intermissions occur in the radial pulse. One of the effects of digitalis is to produce a mild form of heart-block. This, when shown on the tracing, is a delicate evidence that the patient is under the influence of the drug (see Fig. 3).

During partial heart-block there is frequently a dropping-out of ventricular beats at regular intervals. Where every alternate ventricular beat is dropped we speak of two to one heart-block; where every third ventricular beat is dropped we get three auricular pulsations to two ventricular or, shortly, three to two. In the first case the pulse

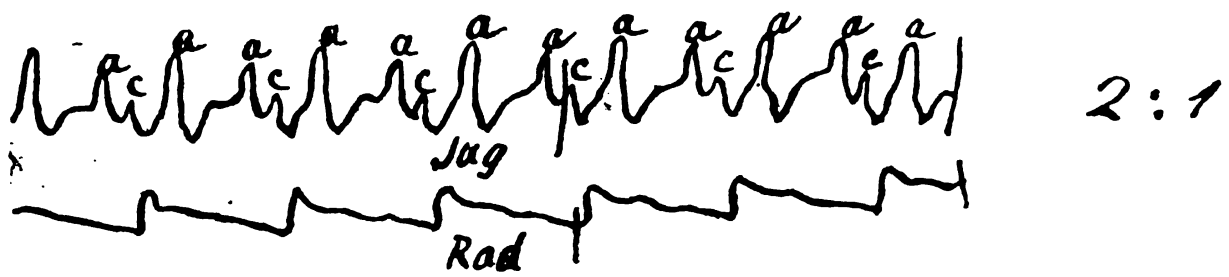

FIG. 4.—"Two to one" heart-block. The ventricle only responds to half the auricular contractions. In the remainder the impulse is "blocked." (Mackenzie.)

FIG. 5.—"Three to two" heart-block. After two normal beats the ventricle fails to respond to the auricular contraction. (Mackenzie.)

would be regular and, as a rule, slow, and in the latter the heart- and pulse-beats would occur in couples. The tracings, Figs. 4 and 5, illustrate these points.

The disease of the muscle-wall on which heart-block depends is often progressive; hence partial heart-block tends to become complete. In complete heart-block there no longer exists any relation between the respective actions of the two chambers. The ventricle takes on a slow action of its own, the so-called "idio-ventricular rhythm." The ventricular rate is generally about 30 per minute, while the auricular is 3 or 4 times more frequent.

The myocardial disease, which causes interruption of the auriculo-ventricular conducting fibres, thus producing heart-block, may be acute or chronic. Myocarditis is exceedingly common in acute rheumatism. We talk of endo- and pericarditis, but are apt to ignore the accompanying myocarditis. Polygraphic observations have demonstrated the occurrence of mild degrees of heart-block during rheumatic attacks, and these have also been noticed in connection with pneumonia, influenza, and other febrile diseases. Syphilis is a rare, but well-established cause of heart-block. Chronic degeneration of the myocardium, such as occurs in the senile heart, is another cause of this disorder.

The milder degrees of heart-block cause no symptoms, except perhaps transient attacks of syncope. Grave symptoms are more common in incomplete than complete heart-block. "Stokes-Adams' disease" is characterised by syncope, unconsciousness, failure of the radial pulse, and sometimes convulsions. Often the first definite attack of heart-block is quite sudden, the patient falling and becoming unconscious. The failure of ventricular action may last from 10 to 15 seconds, during which period oscillations can sometimes be seen in the jugular veins, indicating contractions of the auricles.

The treatment of heart-block is that of the cardiac disease which it complicates. If due to syphilis the usual remedies should be given.

(4) *Irregularity due to auricular fibrillation.*—This form of irregularity is the one most commonly met with in cases of rheumatic heart-disease, especially

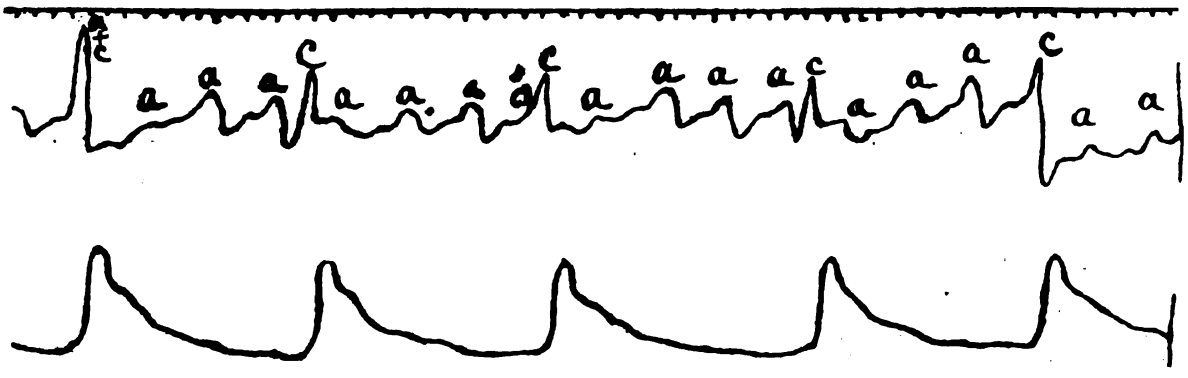

FIG. 6.—Complete heart-block, showing absence of any definite relation between waves *a* and *c*. (Mackenzie.)

mitral stenosis. In this affection the pulse may be exceedingly rapid and irregular. The latter condition may set in suddenly, and may be temporary or permanent. In many cases the irregularity persists until death. In senile hearts the most common form of irregularity is due to premature systole—a condition which comes and goes—but the persistently irregular pulse sometimes seen in this class of cases is due to auricular fibrillation, induced by degenerative changes.

The nature of auricular fibrillation has been elucidated by the labours of Mackenzie and Lewis. It is a particular form of atony of the auricles causing absence of the auricular wave in the polygraphic tracing. There is generally a wave due to pulsation of the jugular vein, but it synchronises with the ventricular contraction, not with the auricular as in a normal tracing. Fibrillation is thus described by Lewis: "The walls of the auricle stand in the diastolic position; systole, either complete or partial, is never accomplished; the wall, as a whole, is stationary, but careful examination of the muscle reveals an extremely active condition; it appears to be alive with movement; rapid, minute, and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface."

In the absence of regular co-ordinated auricular contractions there is also absence of regular ventricular contractions; fibrillation causes irregular and spasmodic stimulation of the auriculo-ventricular band, and from this ensue irregular, disorderly ventricular contractions, generally feeble and rapid —120–140 per minute. Many of the weak pulsations fail to reach the radial artery. Dr. Mackenzie has pointed out that in some cases of fibrillation the pulse is regular and slow. Two associated conditions appear to favour the slowing and the regularity: One is the simultaneous occurrence of heart-block with the fibrillation, and the other is the administration of digitalis.

The occurrence of fibrillation is probably due to fibrotic changes in the auricle, produced partly by the original myocarditis and increased by the strain and tension of the auricular wall. Dr. Mackenzie has pointed out that the presystolic bruit generally met with in mitral stenosis is altered in character after fibrillation sets in. The accented crescendo murmur running up to the first sound is caused by the contraction of the auricle forcing the blood through the narrowed orifice. When the auricle fibrillates the crescendo murmur is lost and is replaced by a diastolic murmur caused, not by the contraction of the auricle, "but by the inrush of blood into the ventricle when the ventricle relaxes after the systole" (Mackenzie). When the heart is beating quickly the diastolic murmur may fill the pause between the second and first sounds; but during the longer pauses, of frequent occurrence in this form of heart disturbance, the murmur falls short of the first sound and does not end with an accentuation, thus differing from the true presystolic bruit (see Fig. 7).

Another sign of fibrillation occasionally met with is the presence of fibrillary waves in the venous tracing during the long pauses of the ventricle. This is shown in Fig. 8.

The presence of auricular fibrillation is an important sign of heart failure, and doubtless greatly aggravates other pre-existing mechanical

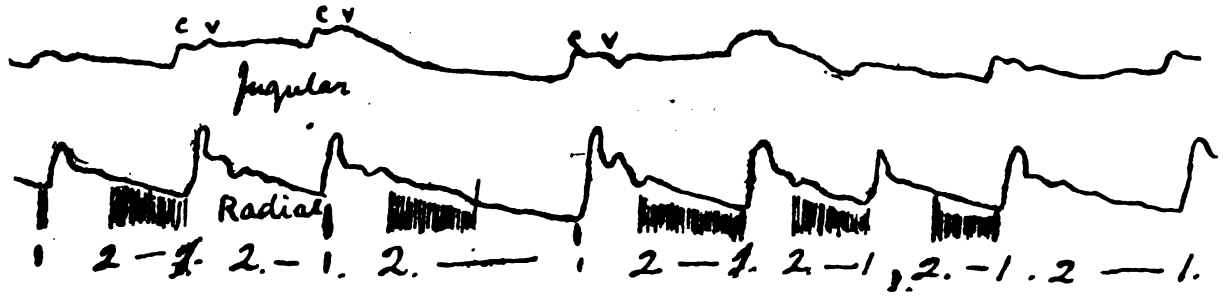

FIG. 7.—Tracing from a patient with auricular fibrillation who had had cerebral embolism. The pulse, at first very rapid and uncountable, improved under digitalis. The tracing shows: (1) that digitalis failed to make the pulse regular; (2) there is no separate auricular wave, as this is merged in the carotid wave; (3) the mitral murmur is diastolic in time, is not "crescendo," and when the pause is long it does not run up to the first sound.

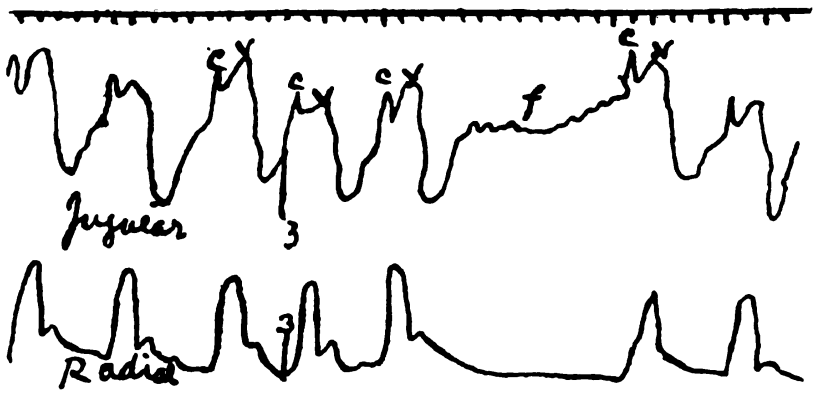

FIG. 8.—Showing at *f* fibrillary waves in the venous pulse during a long pause of the ventricle. (Mackenzie.)

difficulties due to dilatation, valvular defects, etc. Further evidences of failure, such as œdema of the feet or lungs, breathlessness or orthopnœa, enlargement of the liver, etc., may be eventually expected in cases in which fibrillation is persistent.

While digitalis is useful for the purpose of toning up the cardiac muscle in dilatation and many forms of cardiac weakness, it is in cases of fibrillation that results are often achieved which exceed in brilliance those due to the use of the drug in any other circumstances. Doubtless the tonic action of digitalis on the muscle and on the vagus nerve partly accounts for this favourable result, but, considering that the effect is so much greater in fibrillation than in other forms of irregularity, it seems most probable that the more regular and more forcible contractions of the ventricles are due also to the "blocking" action of digitalis, by means of which some of the abnormal impulses from the fibrillating auricles are cut off. In using a powerful drug like digitalis it is necessary to have a clear conception of the conditions it is likely to benefit. There could be no greater mistake than to give it indiscriminately to every patient who has a murmur. This drug should not be given for premature systole, as the disturbance is one digitalis is very liable to produce. It may be given cautiously in heart-block.

The dose of tincture of digitalis which should be given in cases of fibrillation is ℥xv four times a day, or slightly larger doses of the 1914 preparation. In case of urgency the dose may be increased or supplemented by the injection of five minim doses of Hoffmann's digalen. The pulse generally drops from upwards of 100 to about 60 within a week. This is a signal for withdrawing the drug for a few days. It should be resumed as the rate of the pulse increases, and continued in such doses as will keep the pulse-rate about 70. Signs of digitalis sufficiency which call for its suspension or reduction are: Slow pulse; vomiting; loss of appetite; diarrhœa; scanty urine; coupled beats. Cases of sudden death from digitalis are invariably due to neglect of warning signs. Dr. Mackenzie speaks of the "characteristic coupled beats" as a sign of sufficiency, and Dr. Lewis says "the appearance of coupled heart-beats is always a danger sign." The following tracings (Figs. 9 and 10) were taken from a young man whose heart did not show signs of improvement until "coupling" was induced, and in whom small doses of digitalis are now sufficient to maintain this effect without any apparent detriment.

(5) *Other Causes of Irregularity.*

Alternation of the pulse.—By this is meant a series of stronger and weaker heart-beats occurring alternately. This can be seen in pulse tracings, but is rarely perceptible to the finger. It is a sign of unfavourable prognosis, and occurs in degenerated states of the vascular system, such as fibroid heart, arteriosclerosis, and chronic renal disease. It is seen in the failing pulse of acute pneumonia and in many forms of heart exhaustion.

Another form of irregularity met with in pneumonia and other exhausting diseases is due to *failure of contractility*. The whole heart muscle

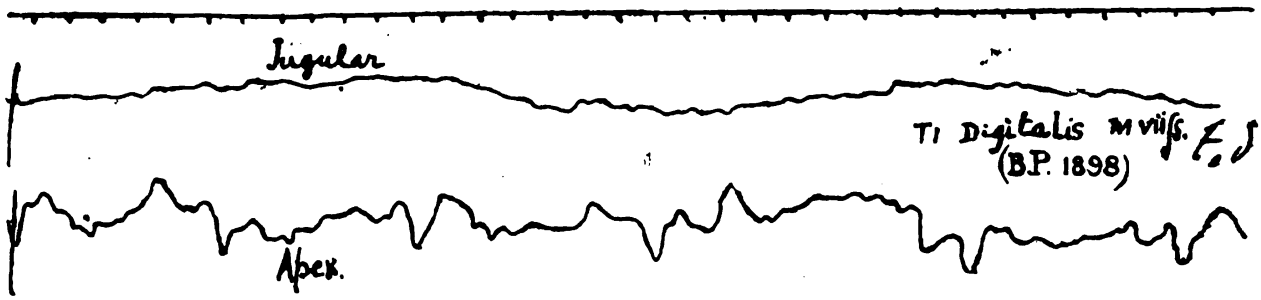

FIG. 9.—Tracings taken soon after admission to hospital from a patient with auricular fibrillation. The upper tracing is from the jugular vein and the lower from the apex of the heart. Both tracings show complete irregularity of the pulse and its feebleness. The doses of digitalis shown on the charts refer to the B.P. 1898 tincture.

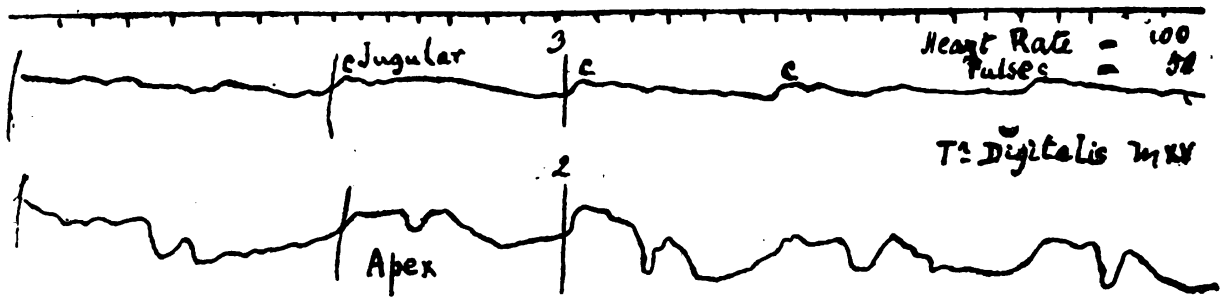

FIG. 10.—Taken from the same patient as Fig. 9, a young man of about 22. The tracing shows a larger carotid pulse and a stronger and more regular apex beat. The beats are coupled, and for this reason the digitalis was discontinued and then resumed in smaller doses. Since last autumn (1914) he has been taking ℳ viiss twice or three times a day, with instructions to leave the medicine off for a few days occasionally. He has been in good health ever since leaving hospital, notwithstanding the fact that his heart-beats are always "coupled," and the radial pulse is half the heart-rate. *Note.*—This condition was still present when he was last seen (July, 1915).

being exhausted, the organ fails to respond to the usual stimulus. This is of ominous prognostic import, and must not be mistaken for premature systole. The latter form of disturbance may also occur, though rarely, in severe febrile affections; but the point to remember is that irregularity occurring in the course of these diseases, especially in their more advanced stages, is generally a sign of serious heart failure.

Most practitioners have seen cases of paroxysmal tachycardia. Their connection with premature systole has already been alluded to. This association was well seen in a woman of about 40 years, who came to hospital a few weeks ago. The pulse-rate was about 160. She said she had had numerous attacks, which came on and went off suddenly. During them she felt unfit for exertion, but did not suffer much distress. She refused to come into the hospital. I saw her again a few weeks later, and the cardiac condition confirmed my opinion that she was subject to attacks of paroxysmal tachycardia, for now the action of the heart, though of normal rate, exhibited frequent premature systoles. A course of bromide, with rest during the attacks, is the best treatment.

When auricular fibrillation is present with tachycardia the pulse is extremely irregular and rapid. Of recent years a variety of this form of disorder has been described under the name *auricular flutter*. The auricles beat with extreme rapidity, as much as 300 per minute, and the ventricles, unable to sustain so rapid an action, may beat at half the rate or still more slowly, as many impulses fail to get through.

November 15th, 1915.

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We regret that owing to the war the number of pages must be diminished for a time.

THE USE OF SKIN GRAFTS IN THE AMBULATORY TREATMENT OF ULCERS.*

By JOHN STAIGE DAVIS, M.D.,

Fellow, American College of Surgery; Instructor in Surgery, Johns Hopkins University, Baltimore.

IN the out-patient department of every large surgical clinic there are many patients with ulcers of varying ætiology, many of them of long standing. The treatment is usually carried out in a routine manner with the idea of getting through with the dressings as quickly as possible. Considering the unsatisfactory results ordinarily obtained, the waste of time and materials is enormous. The wage-earning capacity of the patient is nearly always considerably lowered, and in some instances their work has to be discontinued. It seemed to me that the results might be improved, and the restoration of the full earning capacity hastened if each wound could be studied separately and treated by what we may call "intensive methods."

About two years ago a trial was made of this idea in the Johns Hopkins Dispensary, and I was given the opportunity to study and treat a large number of these wounds. Many old and well-proved methods were used, as well as a number of new, and some hitherto untried, methods. Such favourable results were obtained that special rooms were set aside for this work and every facility given. Among a number of methods for accelerating the healing of ulcers, the *transplantation of skin* was tried on a series of cases.

It has always been taught that the first essential for success in skin transplantation was absolute rest with immobilisation of the part. I fully agree with this principle as the ideal procedure, and

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believe that it should always be carried out when possible. The use of skin grafts as ordinarily done in hospitals on clean wounds, with patients in bed and having the maximum of good food, good nursing, cleanliness, fresh air, and, above all, complete rest, is comparatively simple to the experienced surgeon. On the other hand, take the patients who come to the out-patient department. They are usually poorly nourished; the houses in which they live are often overcrowded and unsanitary, they are, as a rule, unable to stop their work, except for the time taken off for dressings. Many of them should be in the hospital, but there is little chance for even the few who desire admission to secure beds, as there are very few beds available for chronic ulcers. In short, rest, the factor of greatest importance in the treatment of these cases, which is emphasised by every writer on the subject, has to be entirely eliminated. With all these unfavourable conditions in mind, I began gradually to prepare certain wounds with the idea of grafting. Fortunately the first case grafted, that of a long-standing varicose ulcer, was, to my surprise, a complete success, and stimulated further trials.

All of the grafts were placed on undisturbed granulations. I will not discuss at this time the methods used to bring the granulating wounds into a healthy condition; suffice it to say that many operative methods and local applications were used to accomplish this. The technique of cutting, applying and dressing the grafts was that ordinarily used.*

It is obvious that when the grafts are in place they must be secured so that no sliding motion is possible. This is easily done by either placing overlapping strips of rubber protective, or a sheet of paraffined mesh over the grafts and then securing this and the overlying gauze dressing with numerous strips of adhesive plaster. Over this again is placed more gauze and a snug gauze bandage, and finally a muslin or crinoline bandage. Sometimes thin strips of splint wood were incorporated in the dressings. Each patient considered in this series has continued his or her daily occupation.

This report will analyse the results obtained in the first fifty cases in which grafts were used. For convenience the cases may be divided as follows:

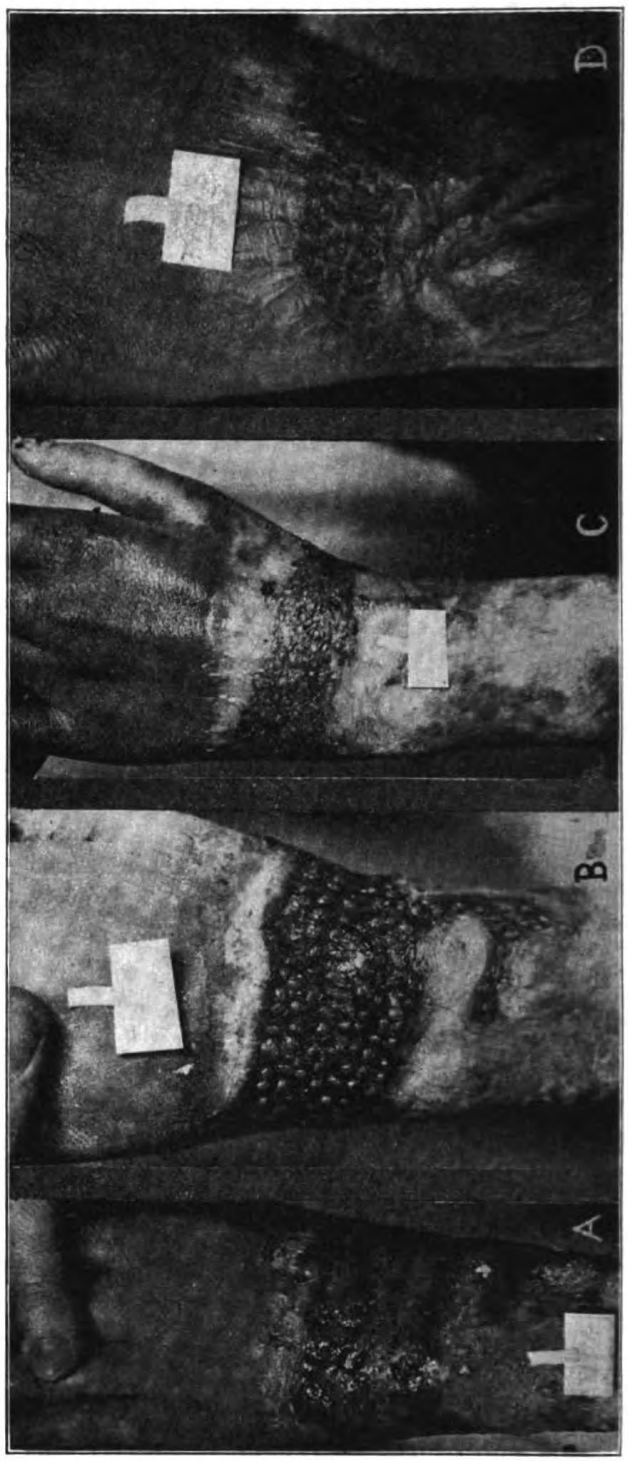

FIG. 1.—Girl, æt. 14 years; ulcer following burn; duration, six weeks. A. Before grafting. B. Two days after grafting with small deep grafts. C. Twenty-six days after grafting. D. Eight months after grafting. This patient has a perfect functional result.

* Davis, J. S.: "Skin Transplantation, with a Review of 550 Cases at the Johns Hopkins Hospital," 'Johns Hopkins Hosp. Rep.,' 1910, xv, 307; "Use of Small Deep Skin Grafts," CLINICAL JOURNAL, October 6th, p. 321.

Summary of Cases.†

(1) Ulcers following burns; 8 cases: Males, 4; females, 4. White, 3; coloured, 5. Ages, 11 to 50 years. Duration: 3 weeks to 22 months. Situation: Leg, 6; cheek and wrist, 1 each. Size: Largest, 16 by 6 cm.; smallest, 2 by 3 cm. Treatment: Small deep grafts were used on 7; and whole thickness on 1. Result: Well, 7; improved, 1.

(2) Ulcers of leg; 9 cases: Males, 3; females, 6. White, 8; coloured, 1. Ages, 39 to 61 years. Ætiology: Six were varicose ulcers with markedly enlarged veins. Three were luetic ulcers with enlarged veins in one. Duration: 3 weeks to 20 years, continuously; off and on, 3 years to 25 years. Situation: Middle third, 2; lower third, 7; right leg, 2; left leg, 6; both legs, 1. Size: Largest, 8 by 17 cm.; smallest, 2·5 by 1·5 cm. Treatment: Small deep grafts were used on all. Results: Well, 6; improved, 2; unimproved, 1.

(3) Ulcers, post-operative; 17 cases: Males, 12;

† Some of the lesions referred to in this report are, according to definition, granulating wounds. To my mind there is a distinction without a difference between a sluggish granulating wound and an ulcer covered with granulations, whatever the ætiology may have been.

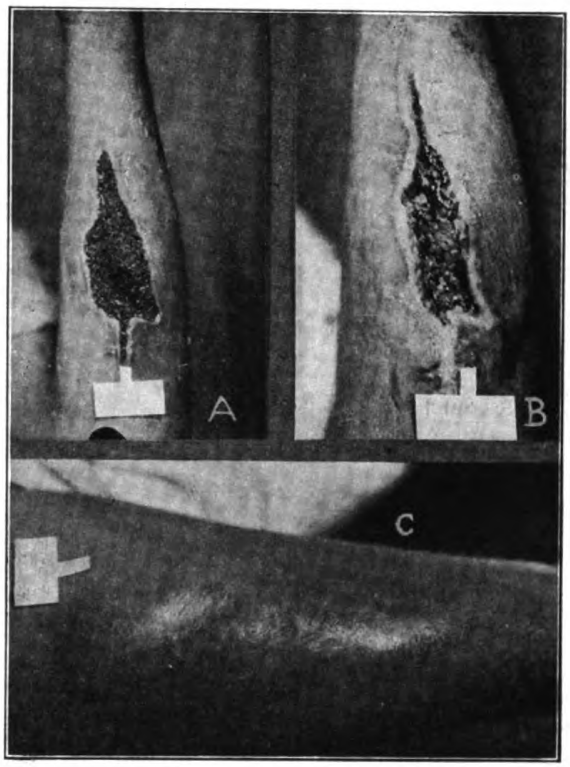

Fig. 2.—Man, æt. 22 years; ulcer following gunshot wound; duration, 4 weeks. A. Before grafting. B. 12 days after grafting with small deep grafts. C. One year after grafting. The skin is freely movable over the grafted area, and the result is excellent.

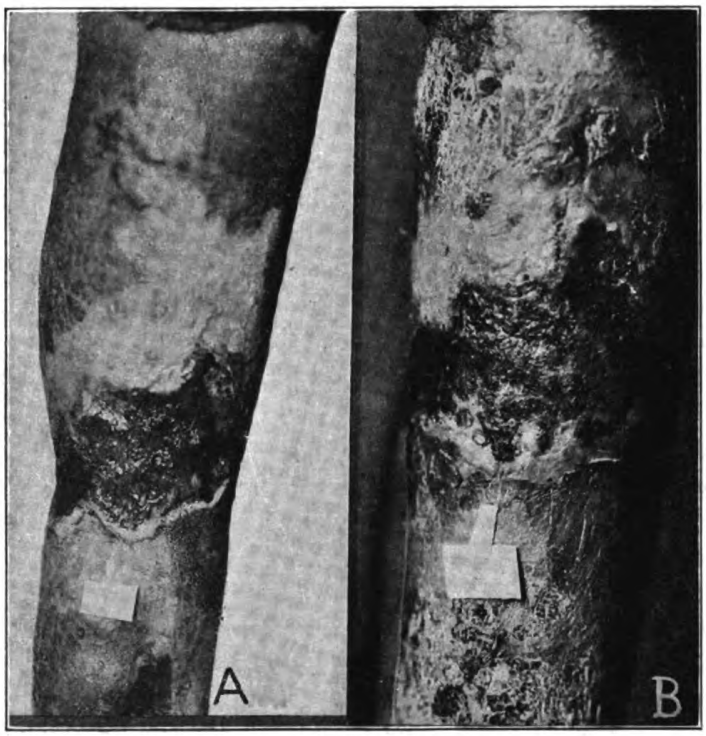

Fig. 3.—Man, æt. 50 years; ulcer following burn; duration, 22 months. A. Before grafting. B. 1½ months after grafting with small deep grafts. Note the solid healing in the midst of scar tissue

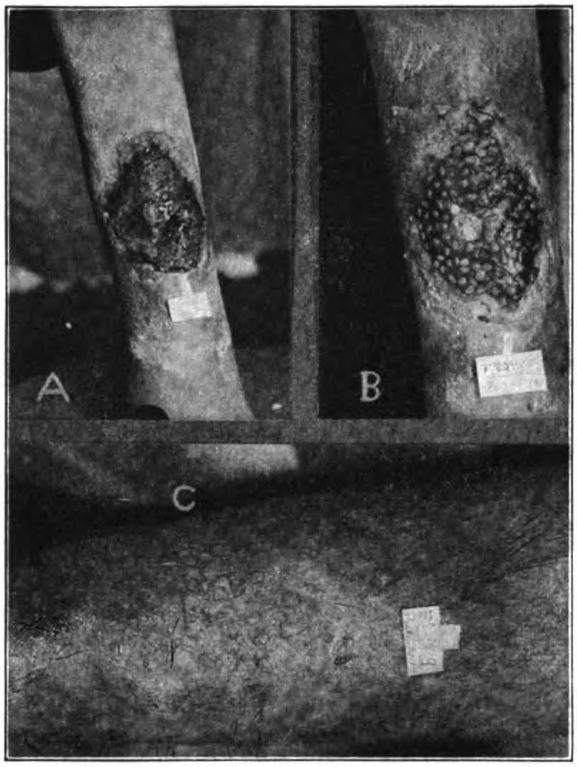

FIG. 4.—Woman, æt. 46 years; ulcer of leg, varicose; duration, 12 years, continuously. A. Before grafting. B. One week after grafting with small deep grafts. C. Five months after grafting. Examination of this patient eleven months after grafting showed no tendency to recurrence. The healing was stable and the individual grafts are still plainly visible.

females, 5. White 16; coloured, 1. Ages, 12 to 84 years. Ætiology: The ulcers followed amputations, operations for ingrown toe-nail, epitheliomas and infections. Duration: 4 days to 3 months. Situation: Finger, cheek, forehead and forearm, 2 each; leg, breast and scalp, 1 each; toe, 6. Size: Largest, 19 by 5·5 cm.; smallest, 3·5 by 2 cm. Treatment: Small deep grafts were used on 16; Thiersch grafts on 1. Result: Well, 14; improved, 3.

(4) Ulcers, traumatic, 9 cases: Males, 7; females, 2. White, 7; coloured, 2. Ages, 11 to 63 years. Ætiology: Injuries of various kinds. Duration: 2 weeks to 10 months. Situation: Finger, 3; leg and ankle, 2 each; foot and wrist, 1 each. Size: Largest, 8 by 6·5 cm.; smallest, 1·5 by 1·5 cm. Treatment: Small deep grafts were used on all. Result: Well, 7; improved, 1; unimproved, 1.

(5) Ulcers, miscellaneous, 7 cases: Males, 5; females, 2. White, 5; coloured, 2. Ages, 13 to 80 years. Ætiology: Following infection and frost-bite, 2 each; trophic, ice bag necrosis, and tuberculosis of chest wall, 1 each. Duration: 2 months to 8 years. Situation: Foot, 4; leg, abdomen and chest wall, 1 each. Size: Largest, 7 by 5 cm.; smallest, 2 by 2 cm. Treatment: Small deep grafts were used on all. Result: Well, 2; improved, 2; unimproved, 3.

General summary: Males, 31; females, 19. White, 39; coloured, 11. Ages, 11 to 84 years. Duration: Few days to 25 years. Size: Largest, 8 by 17 cm.; smallest, 1·5 by 1·5 cm. Treatment: Small deep grafts were used on 48; Thiersch grafts on 1; whole thickness grafts on 1. Result: Well, 36; improved, 9; unimproved, 5. Of those wounds which were improved by grafting but not completely healed, 5 were situated on the foot and 4 on the leg. Of those which were unimproved 2 were on the leg, 2 on the foot and 1 was on the chest wall.

REMARKS.

All of the grafts were autografts. Small deep grafts were used on most of the wounds, as the operative procedure is simple, and, furthermore, no other type of graft could have been successful on many of the wounds grafted.

In some instances, in which the grafts were placed close together, the ulcers were covered with epithelium within a week. When a partial grafting was done, or when only a portion of the grafts were successful, a second grafting was required to fill the areas not covered.

Several graftings were done in all instances of failure, without result. It is interesting that several of these wounds were subsequently grafted in the hospital, without success, so that the failure seemed to be due to the wound itself, rather than to the fact that the patient was not kept at rest.

I feel confident that the percentage of takes would have been considerably larger had the cases been selected as to situation, cause, etc., but in order to test the procedure, ulcers in many situations, and of varying ætiology were grafted. As might be expected, the ulcers on the feet and legs were more difficult to heal than those in other situations, and the failures were almost entirely confined to those regions.

I have been able to observe some of these patients for over a year after grafting, and there has been no recurrence in a single instance in an area successfully grafted. In one or two cases with

marked varicose veins, small ulcers on other portions of the leg have occurred, but not in the grafted area.

CONCLUSIONS.

From the results obtained in the first fifty cases in which grafts were used in the ambulatory treatment of ulcers, I feel that we have added to our armamentarium a method of treatment which has hitherto been used only on patients resident in the hospital. In other words, the successful use of grafts in the out-patient department will make for hospital economy, and will also hasten the return of many patients to full wage-earning capacity.

November 22nd, 1915.

The Ochsner Treatment of Appendicitis.—E S. Hicks states that a visit to the Ochsner clinic in Chicago convinced him that it is a mistake to operate at sight on many cases of appendicitis, and that at times the statement 'that the operation was too late" might be qualified by the addition "or the time for operation badly chosen." Of 200 consecutive cases of appendicitis without a death, 64 were not treated surgically during the acute attack. Operation was performed in 136. Of these 49 were acute cases, operated on as soon as they came under observation—12 were suppurative cases, of which 8 had an abscess drained only, and 4 had both abscess drainage and removal of appendix, while 84 were of the chronic type or were operated after the acute attack had well subsided. The medical treatment, if it can be called such, was prohibition of food, physic, and generally of water. Lavage at times. Rest in bed. Mild heat locally. All patients seen in the first 48 hours were operated on at once if willing, a few were operated on on the third day, but cases from the fourth to ninth days were, especially if very ill, treated medically and a safer time waited for. As the cases were divided between country practice, private hospital and city hospital work, the Ochsner treatment can be commended as a safe and practical outline for guidance even under such varying conditions.—*Canadian Med. Assoc. Journ.*, Sept., p. 767.

A CASE OF NON-DIABETIC ACETONÆMIA,

WITH SOME REMARKS ON ACIDOSIS: ITS CAUSATION AND TREATMENT.

By P. R. COOPER, M.D., B.Sc.Lond., F.R.C.S.Eng.

THIS case is of some rarity, and presented several striking and even dramatic clinical features. The patient was a widow, æt. 26 years, a nurse by profession, who had nursed her husband day and night through a long illness (heart disease), and had then undertaken work in a rather busy district as a district nurse. Here, again, she overtaxed her strength, neglected her meals, and probably also to attend to the calls of nature.

She was fairly well covered, considering her rather slight build, but had a yellow pallor, not of the more usual chlorotic type. She had suffered frequently from "indigestion," *i. e.*, pain after food; flatulence; anorexia; constipation; was short of breath on exertion; had headache, and felt constantly tired, but despite this she had gone on with her work, and had not sought medical advice because she "knew she would be told that she would have to give up her work," and her circumstances, she thought, would not permit it.

However, on getting up one morning she fainted, and as she was alone in her cottage, she lay some time on the floor where she had fallen. On recovering consciousness, she tried to call out to her next door neighbour, but found she had no voice; she succeeded, however, in attracting her neighbour's attention by knocking at the wall, and soon after I was summoned to her bedside.

I found the patient extremely pale, weak and ill. Her temperature was sub-normal (97° F.); the pulse feeble and quick (120); the respirations (30) irregular and frequently sighing; the breath sweet (acetone odour); the voice whispering; the tongue coated with whitish fur, but moist. There was slight complaint of sore throat, and this with the aphonia and breathlessness had given the patient the idea that she was "stricken down with diphtheria." There was, however, no sign of any membrane or even any injection of the fauces, and the absence of any pyrexia soon disposed of this

notion. A careful examination of the chest showed absence of any lung trouble, and the heart-sounds, although quick and feeble, were quite clear, nor was there any apparent cardiac dilatation.

The abdomen was not distended, and although some epigastric tenderness was complained of on deep pressure, this was not marked nor acute. There was complete distaste for food amounting even to nausea, and some tea and milk which her neighbour had given to her had been promptly ejected.

Hardly any urine was passed, and some dull aching in the loins was complained of. There were no catamenia. The urine contained some albumen, but no sugar, and no casts, and gave a strong acetone reaction with Fe_2Cl_6. The bowels were constipated, and had been so for several days.

The patient seemed on the verge of collapse, and there was a certain amount of air-hunger; this, combined with the extreme anæmia and rapid pulse, raised a suspicion of possible internal hæmorrhage, *e. g.* from a gastric ulcer, but no evidence of this was forthcoming. The patient had been very anæmic for some time previously, but had not lost any blood, in fact she had had amenorrhœa. The scanty albuminous urine, aching in loins, headache and sickness, pallor and dyspnœa pointed to possible nephritis, but there was no evidence of œdema, no casts were present, and the acetone odour of the breath favoured acetonæmia rather than uræmia. As the case presented many obscurities, and the patient's condition was rather desperate, I got the late Prof. Dreschfeld to see her with me, and he agreed that the condition was acetonæmia. He could not, however, account for the origin of the condition, and he could not suggest very much in the way of treatment, except citrate of potash internally and carbohydrate feeding. The vomiting for the time precluded the use of these by the mouth, and the first thing was to obtain an action of the bowels. Repeated enemata eventually succeeded in obtaining an evacuation, and rectal feeds of Benger (4 oz.) with ʒj. of potass. citratis were given 4-hourly, all mouth feeding being stopped for 24 hours to give the stomach time to settle. Hot fomentations were applied to the loins, but as the kidneys still refused to act the patient was placed in a warm bath for 20 minutes, and then kept in hot blankets. After 24 hours a little water was retained by the stomach, and the citrate of potash mixture was then given by the mouth. Shortly afterwards stomach feeding with Benger's food, containing a little milk, was cautiously commenced, alternating with an occasional feed of raw meat juice, and the patient began to improve. The condition was critical for a week, and constant nursing was required day and night. As soon as possible an iron tonic was prescribed, and the patient had so far progressed that at the end of a fortnight she was able to be removed to the country for change of air and rest. Apparently all was going well, the patient taking food satisfactorily, the colour improving, the bowels acting well, the amount and quality of urine becoming normal, the strength returning, when I suddenly received a wire to say that a relapse had occurred, and would I go to her at once. Arriving late the same evening I found her even in a worse state of collapse than on the former occasion. The temperature was very sub-normal (95° F.), the pulse 150, she was gasping for breath and complained of great air-hunger, she could not lie down, and insisted upon all the windows being open, although the atmosphere was cold. She was in great distress and felt as if she were dying, and indeed looked very like it also. The collapse had apparently come on fairly suddenly, and without much warning; the only explanation that could be offered was that she had probably walked rather too far for her strength, and may have got somewhat constipated again. (She had been carefully warned not to take any of her tonic unless the bowels had acted well the same morning.)

On this occasion some pain was complained of in the precordial region, but I could not make out any cardiac dilatation. There was again acetone odour of breath, vomiting, and practically suppression of urine—the bladder only containing an ounce or two on catheterisation—this was again albuminous and gave the acetone reaction with ferric chloride. The bowels had not acted for two days, although cascara had been taken at night and a saline draught in the morning.

The patient's condition was so alarming that I deemed it expedient to at once give her $\frac{1}{60}$ gr. strychnine hydrochloride, and $\frac{1}{100}$ gr. digitalin hypodermically, and this seemed to give her some relief. High enemata of warm water containing 1 drachm of sodii bicarb., and 1 oz. of brandy to

the pint were ordered, and hot bottles applied, with some friction of the extremities to restore warmth. I had to spend most of the night and a large part of the next day at the patient's side, and it was necessary to repeat the injections of strychnine and digitalin on three other occasions owing to recurring collapse and dyspnœa (air-hunger). There was never any loss of consciousness, rather restlessness and sleeplessness. The bowel having being cleaned, food enemata were again renewed, with the citrate of potash, and gradually mouth-feeding and citrate of potash mixture were resumed. In about 4 days the patient was again convalescent, breathing normal, urine re-established, bowels acting with the aid of cascara and saline aperients, and appetite returning. I am pleased to add that no further relapse occurred, but the patient was made to take a long rest and strongly cautioned against any undue mental or physical strain. So far as I know she has had no recurrence of the trouble, and has been able to resume her work as a nurse, so evidently she has learnt her lesson in regard to caring for her health.

Now there are many points of interest about this case from the clinical standpoint. Firstly, the whole trouble seemed to be *functional* in character, a disturbance of the *chemistry* of the tissues, without any evident structural or organic change, except perhaps the oligæmia.

The suppression of urine, the albuminuria, the prostration, the dyspnœa, and the vomiting all pointed to a toxæmic condition, the absence of pyrexia contra-indicated an infectious condition, the presence of acetone in the breath and urine pointed to its presence in the blood, and as we know this is usually accompanied by certain closely related fatty acids, especially β-oxybutyric acid, the probable intoxication was the now well-recognised condition of "acidosis."

Acidosis occurs typically in diabetes, where it is the invariable forerunner of coma. It also occurs in a large number of other conditions apart from any glycosuria, *e.g.* in starvation, cancer, anæmia, gastric ulcer and other disturbances of the gastro-intestinal tract, exhausting fevers, and other conditions of impaired nutrition or deficient assimilation. It also occurs in uræmia, the toxæmia of pregnancy, many cachectic states, and after operations and general anæsthetics, especially chloroform. There can be no doubt that the presence of acetone and its associated fatty acids in the blood is not only a grave condition in itself—the acids being highly toxic to the blood and tissues, and interfering with "internal respiration," as shown by the great collapse, anæmia and air-hunger, the emesis, the practical suppression of urine, the albuminuria, etc.—but it also denotes a profound disturbance of metabolism in general. Nitrogenous equilibrium is no longer being maintained, the output of nitrogen greatly exceeds the intake, which may be *nil.* The reserve of carbohydrate (glycogen) in the body is used up, and the nitrogenous tissues have themselves to supply the place of the protein element of the food—the individual is literally living on his own tissues. The disintegration of the tissue proteins results in their separation into a nitrogenous and a non-nitrogenous moiety, and from the latter by a further decomposition are probably formed the fatty acids (aceto-acetic and β-oxybutyric, etc.), which are found in the blood in the condition of acidosis. But whatever be their mode of origin in the blood the presence of these acids can be readily demonstrated. The seriousness of acidosis is quickly appreciated when we remember that one of the essential respiratory functions of the blood is the removal of CO_2 from the tissues. This is ordinarily accomplished by fixing it with the normal alkali of the blood. In acidosis the normal alkali of the blood is neutralised by the abnormal acids present, hence the great dyspnœa or air-hunger, which, however, results not so much from a deficiency of oxygen as from a poisoning with carbon dioxide. For the same reason the relief which promptly follows the intravenous infusion of alkali far exceeds any derived from the inhalation of pure oxygen.

Without going further into the pathogenesis and organic chemistry of the condition, it is sufficient for the present to lay stress upon the importance of the early detection of acidosis in all grave case of illness, especially where there is impaired nutrition of any kind. In diabetes it is a state always to be kept in view and prevented as far as possible. In non-diabetic conditions, however, as in the case quoted above, its results may be hardly less serious. I have no doubt that but for timely treatment this patient would have died of collapse through inanition and acid intoxication. Patients with well-marked acetonæmia who are unable to take food are undoubtedly in jeopardy; they need constant watching and attention. They should be placed in

the charge of a day and a night nurse, and should be under frequent medical supervision. At the least sign of collapse strychnine, digitalin, caffein, and even ether may be required hypodermically. Oxygen, if available, may be of slight service, but the most urgent indication is to neutralise the acid in the blood by means of an alkali. In extreme cases the alkali may be given intravenously. We cannot, however, introduce an unlimited amount of alkali into the blood without causing serious cardiac depression. As a rule not more than 2 or 3 pints of a 6 per cent. solution of sodium bicarbonate, suitably filtered and sterilised, should be given, under strict aseptic conditions, at the temperature of the body, and very slowly admitted —say, taking half to three quarters of an hour for the injection.

Neutralising the acid in the blood does not prevent its formation, however, and to secure this we must endeavour as quickly as possible to re-establish normal metabolism. This can be done only by supplying food, which again can be achieved only by rendering the alimentary canal fit to take in and assimilate food, especially carbohydrate food, for it is found that as soon as carbohydrate food can be absorbed and utilised the nitrogenous excretion greatly diminishes and the acidosis lessens. The indications are to clear the alimentary tract of its impurities and to try to re-establish its secretions. If incessant vomiting is present the stomach tube should be promptly used, and the stomach washed out with sodium bicarbonate solution (one or two drachms to the pint), a few ounces of pure solution being left behind in the stomach.

If the patient is not sick a good purgative should be immediately given, *e.g.* 5 gr. of calomel followed by saline, or 1 oz. ol. ricini, etc. Failing this the bowel should be emptied by enemata, rectal salines combined with sodium bicarbonate can then be given at 4-hourly intervals. As soon as the stomach will retain it an alkaline mixture is given by mouth. Carbohydrate food in an assimilable form is commenced at the earliest moment, and for this purpose Benger's food answered extremely well in the case quoted above. As soon as possible the patient should be got into the open air. Great care in regard to diet, regulation of the bowels, mental and physical effort, etc., must be exercised for a considerable period afterwards to avoid a serious relapse.

November 22nd, 1915.

AMBIDEXTERITY AND MENTAL CULTURE. By H. MACNAUGHTON JONES, M.D., ex-University Professor, Queen's University, Ireland. London: W. Heinemann, 1915. Pp. 102. Price *2s. 6d.*

The book is an amplification of articles which were recently written for 'The Child.' The object is to show that the cultivation of ambidextrous exercises both in art and writing has a decided influence on the mind of the growing child, and, in consequence, on the character. There are seventeen illustrations in the book, some of them done by left hand only, others by simultaneous two-handed writing and models made similarly. The book will appeal to those entrusted in the training and education of the young.

URGENT SYMPTOMS IN MEDICAL PRACTICE. By R. SAUNBY, M.D.Edin., Lt.-Col., R.A.M.C. (T.). London: E. Arnold, 1915. Post 8vo. Pp. 437. *7s. 6d.* net.

If the real and ever-present temptation to symptomatic treatment be avoided Dr. Saunby's book cannot fail to be a most useful and efficient guide, especially to the young practitioner. The subject matter is arranged in alphabetical order, which facilitates reference to the particular disease for which information is desired. Valuable suggestions are made on the management of common ailments, while new and up-to-date matter is represented by disorders like "Flanders foot." Under this heading conditions are described which have been attributed erroneously to frost-bite. The book ought to become popular with the profession.

COMPENDIUM OF THE PHARMACOPŒIAS AND FORMULARIES, OFFICIAL AND UNOFFICIAL. By C. J. S. THOMPSON, Fellow of the Royal Society of Medicine. Fourth edition. London: John Bale, Sons & Danielsson, Ltd. Pp. 360. Price *5s.* net.

The grouping of the official and unofficial formulas in themselves make this book a useful companion. But there is also a large amount of information in a ready and condensed form, including a complete list of synonyms, an index of diseases and remedies, pill excipients, and a synopsis of formulas of the different pharmacopœias—that of the B.P. refers to the 1898 edition. The general practitioner will find this a handy book of reference.

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We regret that owing to the war the number of pages must be diminished for a time.

A CASE OF ABDOMINO-PERINEAL EXCISION OF THE RECTUM.*

By LAWRIE McGAVIN, F.R.C.S.Eng.,

Surgeon to the Dreadnought Hospital and to the Hospital for Women; Lecturer on Clinical Surgery in the London (Post Graduate) School of Clinical Medicine.

GENTLEMEN,—The patient I wish to show you to-day was admitted to my wards six months ago complaining of pain in the rectum, the passage of blood in his motions, and gradually increasing difficulty in defæcation. He is fifty-two years of age, and has never suffered from syphilis or dysentery. On admission he stated that his attention was first drawn to his condition, not by the fact of pain, but that he noticed the presence of small quantities of blood, the motions being coated with it and exhibiting small clots from time to time. For some time he neglected this, attributing it to the presence of piles. As, however, it continued, and he noticed that he was becoming constipated and was losing flesh, and that the act of defæcation was becoming painful he sought advice. On examination it was obvious that the anus was unduly patulous, a condition I have more than once drawn your attention to as suggestive of a stenosis of the rectum. On introducing the finger the lower portion of the rectum was found to be ballooned, and in the second part there was a prominent ring-like mass of growth with a markedly everted edge, the lumen of the bowel at this point being reduced to the diameter of the index finger, which could with difficulty be passed through it. The upper limit of the growth could, however, be reached and proved to be about an inch higher up. The mass could be moved laterally to some extent and the examination resulted in some bleeding, but was only slightly painful. It was obviously a carcinoma, and the question of its removal was discussed by your predecessors with me at the time.

* Lecture delivered at the Dreadnought Hospital.

But I would like to draw your attention to certain points in the case before dealing with the operative treatment. Firstly, I told you that the patient's attention was drawn to his condition not by pain but by bleeding. Now you are probably aware that the public is imbued, most unfortunately, with the idea that cancer is a very painful disease, and so it is many cases, but these are nearly all in an advanced stage. In most cases of cancer of the breast, even when a tumour can be felt, a woman will nearly always ignore her condition if pain is absent, although her sex especially is fully alive to the frequency and the terrible nature of this disease, and will tell you "I did not think anything of it because it was not painful." In the case of the rectum the same unfortunate idea appears to exist, and your patient invariably jumps to the conclusion that bleeding, in the absence of pain, means the presence of piles. Now, gentlemen, I am sorry to say that in this the patient is not always the only one at fault; in the last fifteen years of my experience in the out-patient room of the Hospital for Women, I have again and again come across cases which have been sent to me by members of our profession themselves, with the diagnosis of piles already made, when the patient was the victim of rectal cancer. But what is worse, many of these patients, on being questioned, have told me that their medical man had never made a rectal examination. Two cases particularly occur to my mind in which, having written to the doctors in question with regard to the diagnosis, I received the explanation that the possibility of cancer had not occurred to them owing to the improbability of cancer in patients so comparatively young, one of them being thirty-two and the other thirty-five. Let me urge upon you the duty of making a routine practice of never allowing any patient suffering from rectal hæmorrhage to pass through your hands without the performance of this very important examination.

The second point is with regard to the significance of pain. In the case of piles you will find that pain, as a rule (although not always) precedes the occurrence of hæmorrhage by some appreciable period; further that it is more often troublesome when piles are congested, and obvious at the anus, and is alleviated when eventually the congestion is relieved by bleeding. In the case of cancer the pain is unaffected by the bleeding, and is rather pelvic than anal in position.

Thirdly, the bleeding is commonly free in the case of piles, and only slight in the generality of cases of cancer.

Lastly, in regard to the patulous condition of the anus. The cause of this it twofold, for in the first place Nature will always to some extent deprive us of the use of any organ whose natural function is unexercised for any length of time, whether it be a rested joint, a neglected muscle or group of muscles, or a section of bowel which has been excluded by short circuiting. Consequently the ring-like growth in the rectum having to a great extent taken over the work of the anal sphincters these in time become weak, degenerate and patulous. Moreover, as the growth begins to invade the surrounding parts, the blood supply and the nerve control to these sphincters are interfered with either by direct extension or by a passive œdema, and as a result there is seen the well-known phenomenon of "ballooning" of the rectum and the less noticed patulous condition of the anus.

To return, then, to the case we are examining, the points in favour of attempting the removal of his growth were these: His age was under 60, his condition robust; the position of the growth was favourable, being well removed from the anus and yet within reach of the finger, so that its limits could be clearly ascertained. Its mobility, although a little doubtful, appeared to be fairly free; its fungating character and free hæmorrhage pointed rather to superficial than a deep involvement, and the slight degree of pain and tenderness rather confirmed this. There was no increase in the size of the liver and no enlargement of the sacral glands could be felt.

It was therefore determined to remove the growth, and the question of the best method was discussed. The choice lay between the following:

(1) The direct method *viâ* the perinæum.

(2) Kraske's trans-sacral excision.

(3) The abdomino-perineal excision.

Before discussing any of these methods it is always necessary to consider four other questions, viz.:

(1) Can the sphincters be saved and the bowel reconstituted?

(2) Can the whole lymphatic tract be removed?

(3) Is it wise or necessary to perform a preliminary colostomy?

(4) What are to be the ultimate prospects of the

patient as regards (*a*) his wage-earning capacity, (*b*) his personal comfort, (*c*) his length of life?

The first of these questions is easily answered, and its answer is bound up with that of the second. It is impossible to remove such a growth *so as to include the lymphatic tract and glands* through the limited access afforded by the direct method; nor is it possible, even if attempted, to bring down the upper section *with any degree of success* to the internal sphincter, seeing that such an operation must necessarily be performed through the lumen of the sphincters, to say nothing of the inevitable damage to their nerve supply. The results have been so disappointing from the point of view both of recurrence and of subsequent control that the operation has been justly abandoned.

The question of preliminary colostomy is also easily answered, and answered with regard to all these varieties of operation. Whether the direct, Kraske's, or the abdomino-perineal operation is contemplated, the condition of the patient where no preliminary colostomy is done is a very wretched one. Not only is he persistently worried by the outpouring of fæcal matter, necessitating prolonged and painful cleansings and changes of dressing, but the condition of his wound becomes such that the ultimate results of the operation are to a great extent vitiated if he recovers, which last, owing to pain and suppuration, is quite problematical. I look upon a preliminary colostomy as a *sine qua non* in all three operations.

The last question I would answer as follows. Assuming that his operation, whatever its nature has been, has been preceded by colostomy, and that such colostomy has been conducted in the best manner, his wage-earning capacity should not be interefered with, and that it is not interfered with I know from ample experience.

As regards his personal comfort, this, too, will depend on the efficiency of his colostomy, for although it is unreasonable to expect a man to have the same degree of comfort as before, there is no question that this discomfort can be so minimised that it is amply counterbalanced by the benefit accruing to him by his freedom from pain, sleepless nights, bleeding, and the irritating discharge either from an unoperated cancer, or from an uncolostomised and sphincterless bowel.

Lastly, his prospect of life. This is not so easy to reply to. With regard to the direct method and Kraske's operation it may be said that immunity from recurrence is hardly to be hoped for. Sooner (in the direct) or later (in Kraske's operation) the growth returns, in most cases in the higher glands of the sacral chain, which are difficult of access, or in the liver, etc. The direct method and Kraske's trans-sacral excision are then both imperfect methods of removal, and have led gradually to the third method, viz. the combined abdomino-perineal operation, for it is unreasonable to suppose that free access to the sacral lymphatic chain and glands can be obtained by either of the former operations. If you will examine the patient you will notice first of all that his abdominal wound extends from two inches above the umbilicus to the pubes; secondly that it shows the stretched and rather pigmented appearance of a secondary union cicatrix; thirdly that the anus is absent and is replaced by a puckered cicatrix; and, lastly, that his colostomy wound is placed under the left costal margin and internal to the left semilunar line, indicating that it is the opening of a transverse and not an inguinal colostomy.

The steps of this patient's operation were as follows: A transverse colostomy was first of all performed by Maydl's method. The abdomen was opened and the pelvic colon was delivered, double ligatured with stout chromicised iodised catgut, and divided between these ligatures. The proximal end was then carbolised and invaginated by two circular Lembert sutures of linen thread. Both internal iliac arteries were found and tied, and the meso-sigmoid was divided down to the iliac fossa and gradually stripped away with the whole of its lymphatic track and glands. The rectum in its turn was stripped from the pelvis in like manner as far as the growth which was found to be rather more adherent to the sacrum than was suspected. Next, the peritoneum between the bladder and the rectum was divided transversely and the former was stripped from the latter. The upper end of the detached sigmoid was now carbolised, wrapped up in gauze and dropped into the pelvis, and the abdominal wound was closed.

Finally the patient was placed in the lithotomy position and a circular incision made round the anus and continued back to the tip of the coccyx. The anus was closed by a purse-string suture, and the rectum being separated from prostate in front and the sacrum behind, the whole length of the

rectum and pelvic colon with their lymphatics and glands were removed through the perinæum. A drainage tube was placed in the perineal wound, a few stitches closing the remainder of it, and the patient returned to bed where he was kept for some days in the Fowler position.

The colon was opened at the end of forty-eight hours, the abdominal wound being sealed off by a collodion dressing and the bowel divided at the end of ten days. In spite of the fact that the abdominal wound became infected by *B. coli* and broke down superficially, the patient made an excellent recovery and is to-day as you see him in excellent health.

Now, gentlemen, I wish to criticise adversely one point at least in my conduct of the operation. In place of trusting to pure carbolic acid and the wrapping up of the distal end of the bowel in gauze, I should, immediately after its division, have invaginated it in the same manner as the proximal end. I am sure that this was the cause of the breaking down of the abdominal wound and on another occasion I would certainly not again take the same risk for the sake of gaining a few minutes. I am told by those who are more experienced in this operation than I am myself, for it is not a very frequently performed one as yet, that there is no necessity to tie the internal iliac arteries, but that the sigmoid branch of the inferior mesenteric artery and the superior hæmorrhoidal are the correct vessels to ligate. Personally I rather doubt this advice. The pelvis is richly supplied by blood from the anterior branch of the internal iliac vessels and, as I know from experience, free bleeding may occur here when stripping the rectum, and is very disconcerting and difficult to arrest in a deep pelvis. Of three patients in whom I have recently performed this operation I lost one, and in this case I did not tie these vessels, but did the operation as usually recommended. The bleeding was free and the patient lost much blood, whereas the other two lost almost none.

There is no doubt that, do the operation as we will, it is an extremely severe one, and therefore anything that will tend to diminish shock is to be recommended. The three chief dangers are shock, hæmorrhage, and suppuration. Let us consider them. What else can be done to avoid shock? In the first place the blood-pressure must be raised prior to operation by the administration of pituitrin hypodermically; to get the full effect of this drug a full capsule should be given an hour prior to operation. Infusion of dextrose saline solution on the table will help very greatly if necessary, being given either intravenously in 2·5 per cent. solution, or into both axillæ in 5 per cent. solution. But the greatest factor in the abolition of shock I am sure lies in the use of spinal analgesia so far as it can be used alone, or in conjunction with general anæthesia if it cannot, which may be administered in the form of intravenous ether or possibly hedonal. That this is so was shown in the first of my cases, that of a frail anæmic woman, æt. 67 years. She was nervous, but agreed to the method readily. She was given 6 c.g. of stovaine, her pulse being at the time 120 purely from excitement. The analgesia was pushed to the level of the nipples. The colostomy was performed, and all went well till traction was exerted on the upper part of the rectum during its separation, when very light chloroform anæsthesia was given for forty minutes, the patient never being really unconscious. The perineal excision and remainder of the operation was completed entirely under stovaine, and although the whole proceeding occupied just under two hours the patient left the table with a pulse of 80 and made a perfect recovery, not a sign of shock having been present from first to last.

The question of hæmorrhage I have mentioned. Sepsis is, or may be, a serious matter, owing to the extensive nature of the operation and the large area denuded. It is, of course, most commonly due to *Bacillus coli* infection either from the cut ends not having been perfectly isolated, or at times owing to the unavoidable giving way of a friable growth during separation from the sacrum or prostate. Free drainage, and perhaps the use of *Bacillus coli* vaccine some days prior to operation as a prophylactic are the best safeguards, and, of course, the most careful technique at the operation.

With regard to this latter, I would say a word on the method of dividing the bowel and its subsequent treatment. Three methods present themselves for choice: (*a*) Simple ligation and division; (*b*) clamping, division, and suture, etc.; and (*c*) crushing, ligation, and division, either simple or by the actual cautery. I have employed all three at various times and for various conditions, and after due consideration I am of opinion that the first method is good; the second, although it takes

rather longer, is better, while the third, which is the most recently devised, is rapid, but unscientific and dangerous.

The objection to simple ligation is that a small projection of mucous membrane must protrude beyond the stump, and then being over-sewn by a Lembert's suture leaves a possible source of trouble buried in the abdomen, for it is difficult to disinfect this end of mucous membrane whether by carbolic acid, pure lysol, or the actual cautery. The method is one which I have for years employed with every satisfaction in the case of the appendix, but the stump left in this case is a very small one as compared with that resulting from ligation of the large intestine. Moreover, the thickness of the puckered up mesocolon makes the inversion of the blind end very difficult. The division and suture of the cut ends, since it leaves no dead muscle or mucous membrane in the stump, is clean, scientific, and good surgery, and really involves no serious loss of time, and owing to there being no forcible constriction sloughing is less likely to occur.

The last method, which probably originated in Battle's well-known idea of crushing the stump of the appendix, is, I think, open to severe criticism. The idea that the mucous and muscular layers when crushed by such instruments as the heavy clamps of Robson and Peyer retract in the same manner as does the interior of an artery, curling up and sealing the lumen of the bowel, is erroneous. The mucous membrane and the muscle are not elastic structures like the intima is, nor is the bowel a narrow rigid tube like an artery. Thus, when the bowel has been crushed, the mucous membrane and muscle fibres retracting but slightly, remain as a thin, bruised and probably sloughing ring of tissue, the ready healing of which within the bowel is hardly to be expected. Thus, even when the divided end of the bowel is invaginated, the duty of safeguarding the abdominal cavity is left chiefly to the peritoneum till such time as the two inner coats have cicatrised.

The only two cases of resection of the large intestine which I have lost appeared to me to be due, in one case, to this method of operating, and in the other to simple ligation and division, although the evidence was less conclusive in this latter case.

I feel convinced that the safest and most surgically sound method in these cases is deliberate through-and-through suture, as in pylorectomy, followed by invagination by two circular Lembert sutures.

There are two situations, and only two, from which infection of the tissue is likely to occur, viz., the divided ends of the bowel, and the vicinity of the growth from the tearing of the bowel; especially when it is partially adherent to the front of the sacrum. The first mode of infection can be avoided only by carefully packing off the edges of the wound and seat of division outside the abdomen; avoiding the smearing by swabs which results from careless technique; the carbolisation of the stumps; the discarding of the instruments used in the division and primary sutures; and the final careful cleansing of the invaginated ends by sterilised water or saline lotion. It is certainly not safe to trust to the use of gauze wrapped round the distal portion of the bowel when it is dropped into the pelvis.

With regard to the second mode of infection, it is not always easy to avoid opening the bowel if it is to be stripped off from the sacrum, and it is questionably sound surgery. I am becoming inclined to think that in such a case it would be wise to resect that portion of the bone to which the bowel is adherent. In such a way, not only would infection be less likely, but the risk of recurrence *in situ* would be distinctly lessened. It would, of course, be impracticable to remove more of the sacrum than is the custom in Kraske's operation, but it is just at this point that most of these growths are found.

The danger to the patient is of course far greater in the first than in the second variety of infection. The patient I have showed you was unfortunately the victim of both, but the trouble was not very acute, and he made an excellent recovery.

There is one complication which you must remember in this, and indeed in all forms of excision of the rectum. It is the supervention of incontinence of urine. For a matter of three weeks the patient was unable to hold his water. At the end of that time, however, his treatment by faradisation, strychnine, etc., had the desired effect, and he experienced no further trouble. He has at the present time full urinary control. This incontinence is not difficult to account for. It is due to interference with the fourth sacral nerve

which controls the sphincter vesicæ. It is rather remarkable that permanent incontinence of urine is so seldom seen after excision of the rectum, since the risk of damaging or dividing this nerve must be considerable. It is, however, rare even after the abdomino-perineal operation.

The last point I wish to draw your attention to is the position of this patient's colostomy opening. There are few operations in surgery about which surgeous have been so conservative as colostomy. For some reason or other the inguinal operation has been regarded for many years as the only method worth considering. I will endeavour to show you that the idea is erroneous, and that in many respects the transverse operation which I advocated some years ago is superior. When one deliberately advises such a departure from the recognised methods as this, there must be some good reason for abandoning so time-honoured—I would like to say "hide-bound"—a procedure. Let us first consider the conditions which a good colostomy should fulfil, admitting frankly that nothing can justify the performance of such an operation except the most pressing necessity and the inability to do anything better for the patient.

These conditions I would present to you as follows:

(1) Absence of extravasation.
(2) Freedom from tendency to prolapse.
(3) Ease of access for cleansing.
(4) Position should allow of good belt pressure.
(5) Avoidance of delicate skin and bony prominences.
(6) Removal from the site of primary growth.
(7) Some attempt at sphincter control.
(8) Good "spur" formation.

Now I would ask you to consider how far an inguinal colostomy is capable of providing these desiderata.

The distance between the splenic flexure and the colostomy opening is about 4–5 inches, and the position of the descending colon is vertical. The combined effect of these two factors is that the moment the fæcal mass rounds the corner of the splenic flexure it is acted upon by gravity, and reaches the site of the opening. As the mass accumulates its weight increases, and the opening is subjected to the not inconsiderable pressure of a column of fæces an inch or so in diameter, and 4–5 inches in height. Any form of colostomy opening or belt must be extraordinarily efficient to control the tendency of such a mass to escape. We know the difficulty of avoiding extravasation; it is the chief drawback to this operation.

Now extravasation must be prevented partly by sphincter action, and partly by belt application. But here is a difficulty at once; the material at our disposal is of the poorest, for the making of any sphincter, and the situation is a bad one for carrying a belt. The material is poor in any of the three situations in which the bowel can be brought out, and these are, firstly, through the oblique muscles, secondly, through the linea semilunaris, and thirdly, through the rectus muscle. Owing to the absence of firm aponeurosis *behind* the muscles in the first and last sites, and the presence of aponeurosis *only*, in the second of the three, any steady control of the opening by its circumferential tissues is absent. Again the sigmoid mesocolon is at times of insufficient length to reach the third position, and occasionally traction of the rectal growth may shorten it so that it cannot be brought to the second site with sufficient ease for good spur formation. Nor is this site a good one for a belt, for in women, the width of the pelvis, the prominence of the ilia, the slope of the loins, and the tendency to abdominal obesity in later life prevent the exercise of any great pressure here, and without it no belt and plug will fit snugly. The tendency is always for the belt to ride upwards, and this compels the discomfort of perineal straps. A belt to be efficient in the iliac region needs to be elaborately made, and it is therefore costly and cumbersome. The presence of the iliac spine is a disadvantage, since in elderly and wasted subjects it is difficult to avoid pressure here, and unless this is done the combination of moisture from sweat and extravasation and pressure is apt to result in a very troublesome sore here, the bone even at times being denuded. The skin, too, of this region is often thin and delicate, and not very suitable for the purpose of a colostomy.

Removal of the opening to a distance from the growth, it is true, is not an absolute necessity, but it is desirable at least where the growth is high in the rectum because the mesocolon may be infiltrated and shortened, extravasation of bloody and irritating mucus may occur, and tenderness may be complained of when the belt and plug are applied.

Prolapse of mucous membrane occurs from much

the same cause as extravasation, viz. the action of gravity on a vertical tube. The downward forcing action of defæcation on what is a very loosely attached lining membrane, tends gradually to produce a pouting of the same at the colostomy opening, with at times an actual eversion of all the coats of the bowel, resulting in a moist and excoriated skin. The comfort of the patient will largely depend on the facility with which he can obtain access to the part for personal cleanliness. From this point of view the iliac operation is bad, since in stout patients the bulging abdomen often hides the opening from view, and if extravasation takes place, the groins, which in these people are often affected by intertrigo, become sore and excoriated, and the pubic hair may even be matted with fæces.

The question of providing a more or less efficient sphincter is of first importance, for, although no method has as yet been devised whereby the belt and plug can be entirely dispensed with, some methods are better than others. The ideals to be aimed at are, I think, the following: The new artificial anal canal should lie in muscle of the thickest and stoutest obtainable, and not in fibrous tissue only; it should be supported at its internal and external orifices by aponeurosis; it should be as far as possible supported by neighbouring fixed points; and the greatest care should be taken to avoid damaging the nerve supply of the muscle through which the canal passes.

Attempts have been made to secure sphincter control by dividing the bowel, drawing it through the muscle, and either giving it a half turn before fixing it in the wound, or running it in a horizontal plane through the muscle fibres before bringing it to the surface of the abdomen. Neither of these methods, so far as I am aware, have met with such success as will enable the patient to do without a plug, and the introduction of any such instrument becomes difficult when these operations have been done.

The last point, that of "spur" formation, raises the question as to whether a double opening divided by a "spur," or a single opening made by closing the distal portion of the bowel and fixing the proximal in the wound, is the best. Some excellent authorities are, I know, in favour of the latter method, but I would suggest that for the following reasons the former is sounder surgery: It allows of the distal portion of the bowel being washed out, which is at times necessary and is often comforting to the patient; it is useful for the local application of analgesic, astringent, or hæmostatic drugs to the upper end of a ring of malignant ulceration; and in non-malignant cases it permits of reconstitution of the bowel with greater ease should such fortunate chance arise.

Now, gentlemen, having considered all these points, it remains for us to consider what part of the abdomen or loins affords us the best chance of complying with their requirements.

It is clear, I think, that the usual situation of iliac colostomy does *not* by any means satisfy them. Nor does the lumbar region, which, although long out of date, has recently been again supported by no less an authority than Paul, of Liverpool. Anything more inaccessible to the patient than a lumbar colostomy I cannot imagine. Further, a good spur is most difficult to obtain here, and although Paul would deny its necessity I cannot by any means agree with him.

Now you will notice the situation of the colostomy in the patient before you. It lies in the left hypochondrium, much in the position of a gastrostomy. It is made opposite the end (the *ascending* limb remember) of the transverse colon. I have frequently been asked why I advocate transverse colostomy and why I do it in this situation. I do it deliberately, and as a routine method, and I will give you the reasons.

In the first place, such a situation is most easily accessible to the patient; he can cleanse it and tend the opening without having to bend and strain to do so if he is stout. It is removed from such delicate skin and such a sudorific situation as the groin; and it is at a distance from a growth of the sigmoid or rectum.

In the second place, the bowel here is not affected by gravity, indeed, as it tends rather to turn upwards towards the splenic region gravity would rather act in the *reverse* direction, that is, towards the cæcum: thus extravasation is much less likely to occur.

Thirdly, the mesocolon being of good length there is no drag on the colon, and a good spur can be obtained. In the event (in cases of fistula and non-malignant conditions) of ultimate cure resulting, the reconstitution of the bowel becomes an easy matter as compared with the sigmoid.

Fourthly, a far more efficient sphincter can be obtained here than anywhere else, for if you will consider the anatomy of the part you will remember that here is the thickest, strongest, and most powerfully acting part of the rectus muscle, since it is here close to its bony origin and *point d'appui*. This particular section lies between two of its strong linæ transversæ, and is stoutly backed and fronted by its tough anterior and posterior sheaths. The bowel in its muscular "anal" canal is thus, as it were, moored by "guy-ropes," and this "anal" canal is deeply set between vertical muscle fibres which are always tending to flatten it out.

Lastly, the position is an ideal one for the application of a belt. The abdomen here is, as you see, barrel-shaped, and does not, as in the case of women whose pelves are wide and whose loins are sloping, permit of the slipping upwards of the belt, which compels the use of perineal straps. Pressure is well borne because of the presence of the costal margins, the lineæ albæ and transversæ, and the resistance of the liver and ensiform cartilage in the epigastric angle. These points will, I think, appeal to you, gentlemen, as practical ones. The particular form of belt and plug which I use is constructed, as you see, very simply. The belt consists of a broad band of rubber, perforated like a Martin's bandage and fixed by metal studs. The pad consists of a circular pneumatic bolster surrounding a rubber prominence, which in its turn supports a rubber spur, the plug. A minute tube leads through the centre of these latter, its orifice under the belt being covered by a simple pad of cotton wool, the apical orifice being, of course, in the lumen of the bowel. Thus flatus is permitted to escape through this tube to the comfort of the patient, and the noise of passage of wind between the plug and the skin, which is often a trouble, is avoided. That the operation of transverse colostomy is not a generally accepted one I am fully aware, but my aim is to put before you the *practical* results of clinical experience, and not text-book theories transmitted blindly from generation to generation.

November 29th, 1915.

Hæmaturia from Vesical Varices.—Prof. C. D. Josephson points out that the diagnosis of hæmorrhage from ruptured varicose veins in the bladder is often made; but in women, at any rate, is usually incorrect. When the hæmorrhage is severe, the most common causes are papilloma or cancer of the bladder, or some renal condition. Indeed, he was sceptical as to hæmaturia from vesical hæmorrhoids till he observed the following case. A 4-para, æt. 36 years, had suffered from vesical symptoms, including occasional and insignificant hæmaturia, during the first and second pregnancies. After the second, polypous tissue was excised from the urethra. After her third confinement, which was normal, she developed cystitis. Great difficulty was experienced in catheterism, and the urethra had to be dilated before irrigation with silver nitrate could be undertaken. The cystitis disappeared, although the bacteriuria persisted. Early in 1914 vesical pain and tenesmus were accompanied by urinary obstruction. The patient complained that "something came in the way." There was no hæmaturia. Early in 1915 the vesical symptoms were still troublesome, and micturition was both frequent and painful. Catheterism was difficult, and it seemed as if there was either a urethral stricture or a tumour forming a valve-like obstacle at the internal opening of the urethra. The urethra was dilated, and the bladder was twice irrigated with silver nitrate. Soon a profuse vesical hæmorrhage occurred, and several clots escaped. A week later, when the hæmorrhage had ceased, a retrograde cystoscope revealed to the right of, and above, the internal urethral orifice, small, lobulated prominences, in which blue and distended veins showed just under the mucous membrane. With an ordinary cystoscope these veins would have been missed. The presence of vesical hæmorrhoids was all the more surprising, as the patient did not suffer from hæmorrhoids or varices in other parts. After the cessation of the hæmorrhage a slight attack of cystitis was quickly cured by a few injections of collargol. No local treatment of the varicose veins was attempted, for cystoscopic cauterisation would have been too difficult to perform satisfactorily.—*Hygiea*, p. 1054.

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We regret that owing to the war the number of pages must be diminished for a time.

THE TREATMENT OF SOME COMMON CASUALTIES.

By C. COLGATE HOLMAN, M.B., F.R.C.S.

THE methods described in this article have been employed by me for several years and have given satisfactory results. They are, for the most part, simple and inexpensive.

TREATMENT OF WOUNDS.

The tissues are able to cope successfully with a certain number of micro-organisms. The resisting power is lowered by traumatism. Hence a clean cut wound is more likely to heal by first intention than a contused wound. Foreign bodies, such as ligatures, even though sterile when introduced, tend to delay the healing of an infected wound.

The aim in treatment should be to remove or kill all micro-organisms which have gained access to the wound, and to avoid the introduction of fresh micro-organisms. The latter objective is secured by working with sterile instruments, sterile swabs, and clean hands. The boiling of instruments should present no difficulty. In private practice swabs of cotton wool and squares of plain gauze for dressings can be kept immersed in a solution of carbolic acid 1–20, in covered glass jars. When required for use they will be sterile.

As regards the hands, in theory they should be prepared as carefully for dressing a small wound as for opening a knee-joint. In practice such refinement of asepsis is scarcely obtainable. Provided that the surgeon is careful to avoid contamination of the hands by septic matter, it is sufficient for minor casualties that the hands be well washed with soap and water, and dried on a clean towel.

The cleansing of the wound now demands attention. Some years ago the orthodox treatment was to scrub vigorously the surrounding skin with

ether soap and hot water. The result was that some of the dirt from the skin was transferred to the wound, while the exposed tissues were subjected to a variable amount of trauma. Much of the success of the iodine treatment—at present fashionable—probably depends upon the fact that rough handling of the wound is avoided, while the micro-organisms lurking in the crevices of the surrounding skin are not brought into contact with the tissues.

The following treatment is recommended. If the wound is clean cut it is washed out with a 1–40 carbolic lotion squeezed from a swab. The surrounding skin is gently mopped, or in the case of the scalp, shaved. Interrupted sutures of silk-worm gut or horse-hair are inserted. The wound is then dressed with gauze moistened with 1–40 carbolic lotion and covered by cotton wool.

If the wound is lacerated and dirty, as frequently happens in the case of cycle accidents, the procedure is as follows: The wound is first packed with pledgets of cotton wool soaked in 1–40 carbolic lotion. This controls any oozing of blood and protects the exposed tissues. The surrounding skin is then carefully and gently swabbed and, if necessary, shaved. The packing is now removed from the wound, which is carefully dried, and a diligent search made for particles of gravel and dirt, which are removed by means of dissecting forceps and pledgets of wool. In the case of extensive wounds quite a long time must be spent in ransacking the corners in order to ensure that every visible particle of foreign matter is removed. The wound is next thoroughly washed out with 1–20 carbolic lotion. If the tissues are ingrained with dirt the wound should be lightly painted with undiluted carbolic acid. This will kill the microbes in the bruised and devitalised superficial tissues, and will not penetrate sufficiently to injure the healthy tissue beneath. Carbolic acid acts as a local anæsthetic, and its application is not so painful as that of tincture of iodine. The edges of the wound are approximated by a few sutures, taking care not to tie them tightly. A dressing of gauze moistened with 1–20 carbolic lotion is applied; this ensures against infection of the wound from the surrounding skin.

The bogey of carbolic acid gangrene can be avoided by taking the following precautions. Do not soak the affected part in the lotion. Do not cover the gauze by any waterproof material which delays evaporation. In the case of fingers and toes use only a thin layer of gauze squeezed nearly dry out of a lotion the strength of which does not exceed 1–40.

If the wound is clean cut, the dressings are best left undisturbed for several days or a week, provided the patient is comfortable. In the case of lacerated wounds there is usually considerable oozing of blood, and it is best to change the dressing at the end of 24 hours, if it is hard, dry, and uncomfortable.

A few other details in the treatment of wounds are worth mentioning.

Hæmorrhage can nearly always be arrested by under-running the bleeding points and applying a firm bandage. This method will always suffice in scalp wounds. If a ligature must be used, fine cat-gut is better than silk.

In the case of wounds of the forearm and hand keep a watchful eye for injury to tendons or nerves.

When the dorsal aspect of a finger is wounded it is advisable to apply a splint. It should be fixed by strapping in such a way that the dressing can be changed without disturbing the splint.

Wounds of the leg usually heal by first intention if the patient be kept in bed, by granulation if he is allowed to walk. This is well illustrated in the common case of the small boy who spikes his leg while climbing fences.

Antitetanic serum should be administered in the case of punctured wounds from a garden fork or wounds which have been badly soiled with dirt. The value of antitetanic serum as a prophylactic is generally admitted if it is given soon after the wound is received. Its value when symptoms of tetanus have developed is open to doubt.

Treatment of Scalds.

If the patient arrives with the blebs broken and the underlying surface soiled, it is best to cut away all the detached epidermis and dress daily with a wet dressing of boric lotion or one per cent. aluminium acetate solution.

If fortunate enough to secure the patient with blebs intact, proceed as follows. Carefully cleanse the surface of the blebs and surrounding skin with an antiseptic lotion. Snip each bleb at a dependent spot with sterile scissors and allow the exudate to escape. Wrap the affected part in sterile gauze covered by several layers of absorbent wool, and

keep in place by a firm bandage. At the end of 3 days the dressing is gently removed, any fresh blebs snipped and a fresh dressing applied and left on for 3 or 4 days. If asepsis is maintained the raw surface heals rapidly and painlessly under protection of the detached epithelium.

Removal of Needles embedded in the Hand.

If X rays are available the needle should be localised by screening. Failing X rays an endeavour should be made to ascertain from the patient's statement the direction which the needle has taken in the tissues and the incision for extraction made in that direction from the point of entry.

Before any operation is undertaken the surgeon should refresh his memory of the position of important anatomical structures in the neighbourhood and plan his incision accordingly.

The operation for removal is best carried out under local anæsthesia. Novocain is a safe and efficient drug for the purpose. For small operations solutions of a strength of from 1 to 2 per cent. may be employed. Tablets of novocain and adrenalin can be obtained which yield a solution of suitable strength when dissolved in two drachms of water. A syringe of the "record" type is most suitable for making injections. Syringe, needle, measure, and water must all be sterilised. Care must be taken to avoid the presence of any alkali in the water, as novocain is decomposed by alkalies.

To complete the outfit for operation one requires 2 rubber bandages, 1 about 4 feet long, and the other about 2 feet, a scalpel, a pair of scissors, a pair of dissecting forceps, 2 small retractors, needles, and sutures.

Everything being in readiness the area of operation is painted with tincture of iodine, and the patient asked to lie down on the couch. The needle of the syringe is adjusted with sterile forceps and the operation area thoroughly infiltrated with the novocain solution. The hand is rendered avascular by applying the long bandage from the fingers upwards. The short bandage is applied as a tourniquet above the long one, which is then removed. The part is again painted with tincture of iodine. The surgeon then proceeds to sterilise his hands while the anæsthetic action of the novocain is developing. As soon as the surgeon is ready the operation can be painlessly performed in a perfectly bloodless field. If the subcutaneous fat is very troublesome some of it may be cut away. The edges of the wound may be retracted by an assistant. When the needle has been found the wound is closed, and a pad and firm bandage applied. The tourniquet is then removed.

Removal of a Splinter from under the Nail.

The only reason for mentioning this trivial operation is to draw attention to the value of the ethyl chloride spray as a local anæsthetic in this particular case. The spray is directed on the base of the nail until the whole is white and frozen. With a pair of pointed scissors a V-shaped piece of nail, extending over the whole length of the splinter, can be painlessly cut out. The splinter is then easily picked out with a pair of forceps.

Injuries in the Neighbourhood of the Elbow-Joint in Children.

This type of injury is common in children. Leaving dislocations out of account, the lesion is usually a fracture of the lower end of the humerus, or a separated epiphysis. A description of the various types of fracture does not come within the scope of this article.

The patient is usually brought to the surgeon with a greatly swollen elbow, the forearm being held at an angle of about 120 degrees with the upper arm. In the treatment of this condition many medical men still display a marked affection for an internal angular splint followed by vigorous passive movement. The splint is unnecessary and early passive movement harmful. In children stiffness of the joints following a fracture is usually of short duration, provided the displacement is properly reduced. If active movements were encouraged and passive movements discouraged, we should not so often meet the child who is brought for a second opinion several weeks after receiving an injury to the elbow.

In cases seen soon after the accident it is usually wise to give an anæsthetic, preferably open ether. A careful examination is then made and any displacement corrected. The flexor surface of the limb is sprinkled with a dusting powder and the elbows fully flexed. This position is maintained by strapping the wrist to the upper arm. If there

is much swelling a watch must be kept on the fingers to see that the circulation is maintained.

At the earliest opportunity a skiagram should be taken of the elbow from the lateral aspect, without disturbing the position of the joint. If the position of the parts is satisfactory the treatment is continued.

The fully flexed position is maintained for a period of from 1 to 3 weeks, according to the nature and severity of the injury. If the strapping causes discomfort it may be removed, and the position of the arm maintained by a bandage attached to the wrist and fastened round the neck. As the swelling subsides the forearm is gradually lowered, and the patient encouraged to move his arm a little. As soon as the patient can move his forearm from the horizontal to the fully-flexed position, it should be carried in a sling outside the clothes. In a few more days the sling can be abandoned.

In cases of severe injury it may be several months before the patient can extend his arm fully, but no attempt should be made to force matters.

If stiffness and pain follow a comparatively trivial injury, think of tuberculosis.

December 6th, 1915.

Spontaneous Rupture of Tendons in the Fingers.—Weigeldt reports the case of a carpenter, æt. 26 years, who had been out of work for over a year. Then he was employed in sawing for three days. After his long rest he found it hard work, and noticed that his right wrist had swollen. He kept on working and the swelling subsided, but two days later he had a feeling as if something had broken in his hand. There was little pain, although he could not stretch his thumb. A practitioner treated him by local massage and baths but without relief, and two weeks later a surgeon confirmed the diagnosis of rupture of, and performed a successful plastic operation on, the tendon. In a second case, a woman, æt. 35 years, tore the tendon across in a fall in which the radius was fractured. In a third case the tendon was not entirely severed, but an operation was required. The results in the three cases were excellent.—*Journ. A.M.A.*, p. 1036.

TRANSIENT HÆMATURIA IN CHILDREN.

By ARTHUR J. CLEVELAND, M.D.Lond., M.R.C.P.,
Hon. Physician Jenny Lind Children's Hospital;
Hon. Assistant Physician Norfolk and Norwich Hospital.

Diseases causing hæmaturia are not uncommon in children and are mostly of a serious nature, but I have several times observed an attack which from the clinical standpoint can justly be called transient. I have fairly complete notes of 5 cases in which, for a few days, there was sufficient blood in the urine to be readily detected by the naked eye, which entirely disappeared and did not recur. In 3 children the hæmaturia was not associated with any other evidence of disease, and they have shown by complete recovery and subsequent good health that it produced no permanent ill-effect. The other two were ill during the attack; one has quite recovered, and although the other died, I think it is justifiable to conclude that in his case also the hæmaturia was transient and not connected with his fatal illness.

The first step in the investigation of these cases is to ascertain that the discoloration is due to blood and not to a blood pigment alone, and this can be most readily done by examining the urinary deposit under a microscope for the presence of red blood cells. Having satisfied oneself that blood is present, a most careful examination of the child's general physical condition, as well as of its urinary tract, must be made. If this reveals no abnormality, if the hæmaturia ceases and the urine becomes normal, if the child's general appearance and growth are healthy, it may be concluded that the bleeding has not been due to one of the many organic diseases of which it is usually a symptom.

In briefly discussing the differential diagnosis of these diseases it is convenient to take the general ones first. In the primary blood diseases, in Hodgkin's disease, and in purpura bleeding from the kidneys may occur, just as it does from the gums, or nose, or bowel, or under the skin, but in these and similar states I have never seen the hæmorrhage only in the urine. In infants with scurvy rickets hæmaturia is common, and cases have been described in which it was the only

evidence of the hæmorrhagic tendency, but such instances are very rare, and the clinical picture of the disease is generally well defined. In hæmophilia the bleeding may be from the kidney, and a renal form of this malady has been recorded, but the attacks of bleeding are recurrent, and there is sure to be other evidence of the taint. So, too, in severe toxic conditions, such as the malignant forms of the infectious fevers, jaundice, and "septic" diseases, the hæmaturia is but part of a general tendency to bleed. Renal infarcts are generally found in infective endocarditis, and give rise to hæmaturia, but here, too, other organs will be the seat of embolisms, and the cardiac lesion, the pyrexia, and the increasing severity of the illness will readily indicate its nature. In ordinary valvular disease of the heart I have not often seen hæmaturia, and when I have the cardiac lesion has from the first attracted attention. Poisoning by drugs must be mentioned as a possible cause of the symptom. I have never seen a case, but I imagine there would be other evidence of the accident. In acute and chronic Bright's disease hæmorrhage from the kidney may occur in varying quantity, but I should hesitate to make the diagnosis in the absence of some other general symptoms, such as œdema, sickness, alteration in the pulse tension and heart-sounds, decrease in the quantity of urine passed, and casts in the urine.

Of local causes my experience in Norfolk is that stone is the commonest. A child with a urinary calculus may not give the same well-defined symptoms of it as an adult. So that an X-ray examination of the whole urinary tract must be made if a stone is suspected. Sometimes a calcareous gland may give a shadow suggesting a calculus, and it is not always easy to distinguish the two. A stereoscopic picture will be of great help, as by it one may show that the shadow does not correspond to the line of the ureter. But this may not be sufficient, and it will then be necessary to radiograph with a wire or some X ray opaque fluid in the ureter. In adults what is known as a shower of uric acid or oxalate crystals may give all the symptoms of a true renal calculus, and of this I have met with many instances. The hæmorrhage is, however, slight, the other symptoms much more obvious, and the microscope will readily clear up the diagnosis. In my experience children rarely suffer in this way, and when they do pain is the chief complaint and the hæmaturia not noticed unless specially looked for.

Renal growths, especially sarcomata, are rarely unattended by hæmaturia. Although they generally cause obvious tumours, it is disappointing to find how difficult it may be to ascertain their presence by palpation, and the post-mortem room often affords some unpleasant surprises in this region for the physician. The course of the disease in these cases will soon decide the diagnosis. The progressive failure of the patient and the appearance of secondary deposits will show the malignant nature of the illness. In simple papillomata of the bladder the hæmorrhage rarely ceases for long, and shreds of the growth may be detected microscopically in the urinary sediment.

Invasion of the urinary tract by such an organism as the *Bacillus coli* causes pyuria rather than hæmaturia, and the deposit will be found to be swarming with the bacteria. This condition does not clear up quickly and produces general and local symptoms of considerable severity, and is in some instances a cause of death.

The most difficult factor to eliminate in obscure cases of hæmaturia is tuberculous disease of the kidney. The absence of the bacilli from the urine is not necessarily conclusive. The child's general health may be good, there need not be pyrexia, and the hæmorrhage may be intermittent. Arguing from the analogy of the lungs and knowing the facility with which in some persons tuberculosis can be arrested, it may be held that all these examples of transient hæmaturia in otherwise normal children are really due to tuberculosis. If this is so, then it follows that tuberculous disease of the kidney may in some instances have no clinical importance, and that is a deduction which I am not prepared to accept. One can imagine that in some cases of renal tuberculosis hæmorrhage might be the first and for some time the only symptom, but even if it ceased temporarily, it would not be for long, and other evidences of the disease would soon follow. Similarly, if the bleeding were due to some vascular abnormality, recurrences should occur even if the child's health did not suffer.

In 3 of my cases the hæmaturia was unattended by any constitutional disturbance. In the other 2 there was evidence of an acute illness. In Case 4 the child had been quite well until two days before

admission, when the hæmaturia was first noticed, and was then feverish, irritable, and occasionally sick. She got quite well in 2 or 3 days and never had any of the other symptoms of Bright's disease. Case 5 was especially interesting, as the boy evidently had 2 distinct diseases: the first an acute febrile one affecting chiefly the lungs and associated with marked hæmaturia, and the second a general tuberculosis, which developed after the symptoms of the first had quite cleared up and when the child was apparently well. At the necropsy the kidneys were quite normal in appearance, and no tubercles were present in them, so that I think the hæmaturia was not connected with his tuberculosis, but was due, as in Case 3, to an acute congestion of the kidneys of a transient character. This may be classed as an acute nephritis similar to that which so frequently accompanies fevers, but it is quite distinct from Bright's disease, and is pathologically of a transient character.

I have come to the conclusion, therefore, that transient hæmaturia does occur in children and that, whatever may be the cause, the hæmorrhage itself is of no great significance.

Case 1.—Agnes C—, æt. 8 years. Admitted into the Jenny Lind Hospital, July 11th, 1910. Parents healthy; no family disease. Has always been rather delicate. Whooping-cough when three months old and rheumatic fever (?) when aged two years. For the last month her mother has observed intermittent attacks of hæmaturia. During the attack the child goes off her food, but does not complain of pain, and otherwise is well. Urine contains a lot of blood and albumin; no "gravel" noticed. X-ray examination shows no stone. Child not robust, but organically sound. Temperature normal. Has never had similar attacks before.

The hæmaturia and albuminuria entirely cleared up, and she went out on July 29th, 1910.

July, 1915.—Her mother reports that she has never had any return of the bleeding and is in good health.

Case 2.—Emily W—, æt. 10 years. Admitted into the Jenny Lind Hospital, October 10th, 1913. Consumption on father's side. She had scarlet fever and diphtheria two years ago. Has never been strong. Said to have vomited blood 5 weeks ago. For 10 days before admission had had pain in the left side. Three days ago water was noticed to be dark and has been getting darker. Condition on examination: Thin child, no abdominal tenderness. No pain. Heart and lungs normal. Urine contains blood and albumin. X-ray examination shows no stone.

Dr. Claridge reported that no organisms could be found in the urine. Number of ounces of urine passed in 24 hours, 26–34.

Three days after admission had an attack of abdominal pain, but this was not followed by hæmaturia, which did not recur, and the child was discharged on November 7th, 1913, apparently quite well.

She was readmitted in 1915 for "debility," and sent to a convalescent home. She has had no return of the hæmaturia or pain in the side and is apparently well.

Case 3.—Ernest T—, æt. 9 years. Admitted into the Jenny Lind Hospital, October 18th, 1913. Family history good. Always been healthy. Had a sore throat 3 weeks ago, but no doctor was consulted. For a week before admission the urine has been dark.

A healthy-looking child. Urine contains albumin and blood. Temperature normal. Quantity of urine in 24 hours, 24–38 oz. October 28th: Urine sp. g. 1026, acid, a trace of albumin. No casts. No blood by chemical tests. On November 5th he went out quite well.

July, 1915.—Has been quite well since leaving hospital, and has never had any more bleeding. He looks strong and healthy.

Case 4.—Elsie S—, æt. 3 years. Admitted into the Jenny Lind Hospital on May 19th, 1915. One sister has a tubercular knee. Father and mother alive and well. She was quite well until two days ago, when she was noticed to be passing blood in her urine, was sick at times and off her food.

Temperature 102° F. Tongue furred. Throat normal. Vomits occasionally, and one vomit was slightly blood-streaked. Urine dark brown in colour and contained albumin. No physical signs in chest or abdomen. Microscopically blood corpuscles found in urine. No casts. X-ray examination showed no stone. Bacteriological examination of a catheter specimen of the urine showed no organisms. Child's temperature reached normal in 3 days, and she went out quite well, without any blood or albumin in the urine.

CASE 5.—George M—, æt. 8 years. Admitted into the Jenny Lind Hospital, November 6th, 1913.

He caught cold 5 days before at the funeral of his father, who died of acute bronchitis. He was a strong, healthy man; his illness was acute, and apparently influenza. A necropsy showed acute capillary bronchitis. Other organs healthy. No hereditary disease in the family.

Temperature 101° F., respiration 52, pulse 130. Impaired resonance and diminished breath sounds at right base. Slight cough. Heart and abdomen normal. Urine 1024. Blood present in quantity. Microscopically, blood cells. November 8th: temperature normal. Nothing abnormal to be heard in the chest. Urine 48 oz. in 24 hours. Blood and albumin present. November 11th: Dr. Claridge reported urine sp. gr. 1020. Albumin, a trace. Blood present. Urea, 8 grains to the ounce. Deposit consists of red blood corpuscles, with cellular and granular casts. No organism present. He continued to improve, and his temperature remained normal until December 1st. Urine contained a trace of albumin, but no blood noticed.

From December 1st his pyrexia continued and increased, and he grew steadily worse until death, which occurred on February 20th, 1914. Widal's reaction was negative, and by the middle of the month a diagnosis of general tuberculosis was made.

At the necropsy general miliary tuberculosis was found. There was a large caseous area in the spleen and a caseating patch at the apex of the right upper lobe of lung. Tubercles on the meninges. The kidneys were normal.

December 6th, 1915.

THE illustrations of Mr. Muirhead Little's paper on "Pseudo-Coxalgia" (CLINICAL JOURNAL, November 10th, p. 361) were from X-ray plates made by Dr. Stanley Melville, Radiographer to the Royal National Orthopædic Hospital.

THE TREATMENT OF WOUNDS WITH THE FUMES OF IODINE, WITH SPECIAL REFERENCE TO DEEP WOUNDS.

W. L. CAPELL, M.D.,
Omaha, Nebr., U.S.A., and
JOHN C. ROMMEL, M.D.,
Philadelphia, Pa., U.S.A.

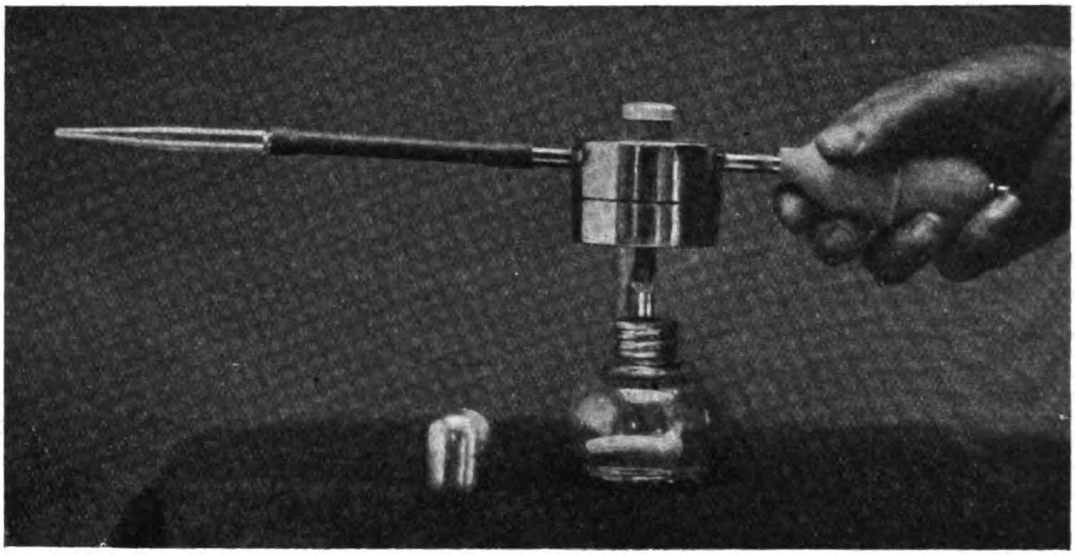
Now that the military conflict is so universal the treatment of the wounds of battle is an ever-present problem. We describe a method of using iodine in a vaporous state that has served us well for a number of years. The great value of iodine in medicine and surgery is being recognised more and more. While we have used iodine in nose, throat, ear and vaginal catarrh and other conditions in those organs in the manner we are about to describe, yet we will here write only of the remedy for traumatic cases.

Every wound, however slight, is likely to become infected, and especially gunshot and stab wounds. Hence, such a wound is always a serious matter, fraught with grave potentialities, and the necessity for the most efficient means to overcome the dangers.

Gunshot and bayonet wounds are usually deep, and there is the danger of bits of clothing-thread, dust, germs, etc., being carried into the tissues. Hence, it is necessary to keep these wounds open at the top until the bottom heals aseptically. Should the integument heal, confining in the deeper part of the wound some of the foreign material, broken down tissue, or pus, repair is arrested by retention of this material.

Another source of danger is that the canal of deep wounds, however straight when made, is prone to develop pockets here and there, or the normal relations of the parts may be such that, after the wound is made, the tissues in recovering may change considerably the course of the wound. The loss of adipose tissue also accounts sometimes for such changes.

The lumen of a tortuous canal, is exceedingly difficult to reach with an antiseptic, either fluid or powder. We have found that iodine vapour under slight pressure answers the purpose admirably. Iodine volatilises at from 120° to 130° F. Re-



sublimation occurs on its coming in contact with a colder surface or colder temperature. The crystals can be vaporised or fumed and in this state carried into the deeper parts of the wound. This method has many advantages, prominent among which is that it is applied warm. Hence it does not affect sensitive nerves, which would cause muscular contraction, partially or wholly closing the wound cavity when we are applying an antiseptic. When iodine is irritating, this is due to the presence of large particles of the remedy. This is well recognised in the making of iodine ointments. Fuming the iodine produces a fine state of subdivision that permits of ready absorption with a minimum of irritation—usually with none.

In the treatment of all infected wounds the frequent application of the antiseptic is important to kill the invading micro-organisms as they mature, for it is questionable whether any known applicable antiseptic will destroy spores. Therefore, if the infection is to be overcome, the bacteria must be destroyed as quickly as they develop. Hence, an unirritating antiseptic must be used constantly.

Another advantage of iodine fumes in the treatment of wounds is that the toxic effects of the medicament may be avoided. Rapid absorption and elimination prevent any cumulative action and the amount used can be definitely controlled. Thus, if only one grain is put into the fumigator no greater amount than that will be used and absorbed.

In the light of these facts, it would seem that the fumes of iodine have a decided advantage over any other form of iodine medication in the treatment of wounds of all kinds, but especially in gunshot and bayonet wounds, that are likely to be deep and narrow. The frequent introduction of the nozzle of the fumigator into entrance of the wound prevents tegumentary cicatrisation until the deeper parts are healed and renders unnecessary the tampon or drainage tube.

The fumigator consists of a metal chamber with three openings (Fig.). One opening is in the top, and the other two on opposite sides near the top. The opening in the top is for the purpose of inserting the iodine and is closed with a cork. From the side openings project tubes. To one is connected a rubber tube and bulb to supply air pressure, as in an atomiser. To the other tube is connected a rubber tube to which is attached a glass nozzle coming to a narrow point. This point is inserted into the wound and the iodine is forced into the wound by compressing the bulb. The iodine is vaporised by an alcohol lamp under the fumigator.

December 6th, 1915.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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We regret that owing to the war the number of pages must be diminished for a time.

FOUR HEART CASES: A CLINICAL STUDY.

By C. EDGAR LEA, M.D.Vict., M.R.C.P.Lond.,
Honorary Physician and Physician-in-Charge of the Cardiac Department, Ancoats Hospital, Manchester; Assistant Medical Officer, Manchester Royal Infirmary.

IT happens that I have in hospital four heart cases which present certain clinical points of interest and contrast. They illustrate the value of viewing every case in its widest aspects, and also they indicate the use of the more modern methods of examination.

CASE 1.—*Mitral stenosis. General visceroptosis.*

M. J—, an unmarried woman, æt. 31 years, delicate from early girlhood, tall, thin, and fragile in appearance, complained of pain over the heart, palpitation, and headache on my first seeing her in February, 1915.

History.—As early as the age of 14 both she and her over-sympathetic mother were told by their doctor that the "valves of the heart were wrong"—a baneful influence from the start. She had not had rheumatic fever. From the age of 7 she grew very quickly, and is now unusually tall. As a child she is said to have been subject to faintings. Seven years ago she had slight hæmoptysis. For many years now she has played the part of the interesting invalid. Occasional præcordial pains, palpitation, headaches, nausea, flatulence, cough, feelings of general fatigue for which she would readily take to her bed—all the usual signs to be expected in a heart case. I suggest, however, that the greater part of this patient's ills was referable less to the heart than to her general condition.

Examination.—(*a*) General: The patient was thin to a degree, the musculature weak. A marked kyphosis, especially in the upper dorsal region, gave a prominent forward pose of the head, which emphasised the general tired expression. Her

mental attitude suggested: "Oh, here's another doctor. He can do no more for me than all the rest." She was not, however, at all of the hysterical type. The heart's dulness was less extensive than normal. The usual murmurs typical of mitral stenosis were present. The chest was remarkably long and narrow, the rib spaces wide, and their general inclination in their lower parts almost vertical. The abdomen bulged in the infraumbilical portion on standing. The lungs were clear. No albuminuria.

After admission to hospital further observations were made.

(*b*) Special examination: X rays confirmed the diminished cardiac dulness, the heart having the appearance, well known in rœntgenology, of hanging, as it were, in its pericardial sac from above (the hanging heart of Wenckebach). Its shadow was prolonged vertically and narrowed laterally. The diaphragm was low, its movement good. A barium meal showed either a ptosed or a markedly atonic stomach, its greater curvature lying almost in the pelvis. The transverse colon was, similarly, in a low position. Electrocardiograph: Normal curve except for the rather prominent "P" wave due to auricular systole and typical of mitral stenosis. Some variation in heart-rate with respiration was noted, but not at all times. Rhythm regular. Rate generally rather quick. Pharmacological tests: The work of Eppinger and Hess have shown that in certain individuals, "vagotonics," the vagus is unduly susceptible and may induce symptoms in the many viscera which it supplies. In these cases it is found that stimulation of this nerve, as by giving jaborandi, may evoke these vagal symptoms, and, conversely, atropine may lessen them. Both these drugs were tried, given by mouth over a certain number of days, but with no detectable effect. This would suggest that vagotonia was not the dominant cause of general symptoms in this case. Blood-pressure: Lying down, systolic pressure 140 mm. Hg., diastolic 85; standing up, 140 and 90 respectively, the pulse-rate somewhat increasing. After a walk, systolic pressure 138, diastolic 100. Tight abdominal pressure, however, with the patient standing, raised the systolic pressure eight mm.

Course.—During her stay in hospital three features were noted: The pulse-rate was very labile, varying on different days from 100 to 120 for no apparent reason; there was unusual variation in her weight; and on one occasion there was definite albuminuria which lasted for only a day or two.

Treatment.—Treatment was directed more to the general visceroptosis than to the heart, which in all probability was in a very fair condition. The chief difficulty was the low nervous tone, the complete lack of the "push and go" element. Diet was liberal; meat being preferable to the patient, who had developed many fads, was allowed, and fatty foods encouraged. Aperients were restricted, enemas being given every third day. Abdominal massage once a day, and abdominal exercises night and morning. An abdominal belt was ordered, which at once made the patient more comfortable than she had been for a very long time. She was ordered also to sit well up in her chair and to resist the lolling attitudes she had got into. Considerable improvement in her general condition resulted, and she increased 7 lb. in weight.

CASE 2.—*Mitral stenosis. Nephritis.*

The patient, E. B—, was a well-developed woman, æt. 45 years. She has been an old sufferer from heart disease. At 20 she had rheumatic fever, and dyspnœa has been present, on occasion, ever since. She has had three children, but has been generally worse since her last born, 13 years ago. An increasing tendency to bronchitis and asthma showed itself; œdema was noted sometimes; and lately palpitation has been especially marked. This would come on with effort, often associated with dyspnœa, but it would also occur even when the patient was at rest. With it is a feeling of "jumping" in the eyes and a fulness in the throat. Dyspnœa was of two forms—with effort and also in paroxysms. Pain occurred, sometimes of a gnawing type, between the shoulders. Frequency of micturition had been noted. Headaches were frequent, limited to the right frontal region, and with it—an interesting feature, noted in other cases—there was often an abrupt watery discharge down the right nostril. Lately she had had gastric symptoms, and for this all her teeth were removed, though with not a great deal of success in so far as these symptoms were concerned. Seven months ago she had herpes zoster in the back.

Condition on admission.—The patient looks fairly well. There is no urgent dyspnœa. The heart's dulness is normal laterally, but rather low, reaching only up to the fourth rib. A well-marked

presystolic and diastolic murmur at the apex confirm the diagnosis of mitral stenosis. The pulse is slow, 60, regular. Lungs clear except for a few *râles* in the low axillary regions. No œdema. Albumin in the urine. Discs normal.

Special examination.—The electrocardiograph showed the well marked "P" wave to be expected in mitral stenosis, due to the vigorous auricular systole. In addition, however, there were fairly frequent extra-systoles arising in the auricle. These had not previously been found. The X rays showed a low diaphragm, the movement of which, however, was good. There was some gastroptosis with active peristalsis. Blood-pressure for a case of this kind was high. Systolic 178 mm., diastolic 88 mm. A tight bandage applied around the abdomen failed to raise the systolic pressure. Pharmacological tests: Belladonna evoked no appreciable change. The eyes, however, were rather susceptible. Jaborandi caused some "burning" sensation in the head and occasional flushes. These she had been subject to before admission.

The general diagnosis of mitral stenosis was, of course, apparent, and it was considered that the majority of the symptoms were indirectly due to this. Unlike Case 1, general visceroptosis could not reasonably be held to be immediately responsible for the dyspnœa and general weakness. Nor was vagotonia very evident in spite of the slow pulse and the slight jaborandi response. This case illustrates, in the variety of symptoms and their irresponsible incidence, the wide range of subjective symptoms to which a cardiac condition can so often give rise. It is, however, when we try to explain their mechanism that difficulty arises. The real obscurity of their genesis ought to be fully recognised, their presence is familiar enough. To those who believe in a heightened irritability of those segments of the spinal cord subserving the nervous mechanism associated with the heart being mainly responsible, some support is in this case offered them from the fact of the herpes from which the patient suffered, and which indicates a focal disturbance in the region of the cord, but the notes do not say at what level this occurred. A diagnosis of interstitial nephritis was made on the basis of four facts. (1) The presence of albumin. This, in a heart case, is not, of course, unusual, and may be secondary to this organ. When, however, it is present and the pulse is regular and within normal limits of frequency, as was the case here, nephritis is almost certain. (2) The frequency of micturition. (3) The dyspnœa of paroxysmal type. This may occur also in some cardiac cases, but it is not usual in the absence of gross rhythmic disorders; such dyspnœa may also, in this patient, have been held to include her so-called asthmatic attacks. And (4) the high blood-pressure. There remained another interesting feature in this case—the palpitation. This is not, of course, an unusual symptom. With the detection of the auricular extra-systoles, however, these attacks assumed an added significance. It is known that they may give rise to palpitation either when, as in this case, arising singly or when arising successively and thus constituting a true paroxysmal tachycardia. It was thought, then, that these extra-systoles were the source of the palpitation, and an opportunity was sought for to examine the patient with the electrocardiograph during such a paroxysm of palpitation. The chance at last arrived and the patient was hurriedly connected up to the instrument, but to our surprise there was no arrhythmia, only a general quickening of the normal rate. It was remembered, too, that when we had previously found the auricular extra-systoles the patient had had no symptoms when they had been present. We must conclude therefore, that, even in a patient prone to cardiac arrhythmias, subjective symptoms may not necessarily be related thereto. But from another point of view these auricular disturbances have significance. They are admittedly freely met with in the stages preceding the passing of the auricles into a state of fibrillation; they may represent, as it were, the early heralding of graver disorders. In mitral stenosis fibrillation is especially common, and from the point of prognosis, apart from the immediate subjective symptoms, these subjectively harmless auricular extra-systoles indicated more than a passing chance that in this patient fibrillation is liable to ensue. This bears also on treatment. Digitalis increases the rhythmic activity of the heart. It may, in fact, itself induce fibrillation. To give this drug in such a case is to run some risk of intensifying the cardiac irritability and sending the auricles into fibrillation.

Treatment.—An attempt was made to widen the circulatory field and to lessen the strain upon the heart suggested by the high blood-pressure by a

course of Schott exercises and home-made Nauheim baths. These extended over a month. The results were satisfactory. The blood-pressure fell to an average of 140 mm., a drop of over 30 degrees, and though repeated electrical examinations have been made in no instance have any abnormal auricular contractions been noted. The general condition is somewhat better, and the albumin quickly disappeared. No digitalis was given for reasons stated. The palpitation, however, has not been much relieved.

CASE 3.—*Rheumatic myocarditis. Cardiac dilatation.*

J. H—, a boy, æt. 12 years, was admitted to hospital on June 23rd looking very poorly. His mother said that he had had rheumatic fever six weeks previously and had never recovered from its effects. Previous to this infection he had had good health. The family history was good and showed no other sufferers from rheumatism.

Examination.—The boy was rather thin, the breathing was rapid, 28, but not laboured. The pulse was quick, 128, regular, and rather compressible. The heart showed especially an extension of dulness to the right; it extended 2 in. from the midsternal line. The nipple line, 3½ in. from the midline, showed the left border. Auscultation revealed a markedly accentuated first sound; the second sound was indistinct, and the interval between it and the next succeeding first sound was filled up by a loud murmur which could be taken to represent a diastolic murmur and, possibly, a presystolic as well. When the rate exceeds 120 it is well nigh impossible to dissociate such murmurs. Over the tricuspid area a soft systolic murmur could be heard. A systolic murmur was audible also in the back, on both sides of the vertebral column. Lungs: Over a vague area below the left scapula harsh breathing could be heard, and pectoriloquy was present over this area too. No moist sounds. There was no cough. No albumin in the urine. Electrical curves showed nothing abnormal.

Progress.—Quite uneventful. In 14 days the pulmonary signs had gone. In 22 days the heart's dulness had lessened to 1¼ in. to the right. Throughout, after the first day or two, the lad was apparently quite well and cheerful, taking his food well, etc. At no time had he any pain. Clinical interest, then, does not centre on the symptomatic features of the case. But it is just this very absence of symptoms which, taken in conjunction with the condition we believe to be present here, namely, dilatation, desires to be emphasised if we are to arrive at a wise practical opinion upon the prognosis and treatment. Upon what is the diagnosis of dilatation based? Mainly upon three findings: (*a*) Extension of cardiac dulness to a considerable degree to the right; (*b*) sustained rapidity of the pulse-rate (throughout the whole time, although this boy was kept in bed, the pulse-rate never fell below 100); and (*c*) general evidences of passive congestion; these in this case were absent. It is important before establishing such a diagnosis to be reasonably sure that the high pulse-rate is not due to any other demonstrable causes, and in this particular case, although I think that the diagnosis of dilatation is correct, there was one factor which must compel me to "hedge" by offering an alternative diagnosis of subacute vegetative endocarditis. I do so for this reason: for the first three weeks after his admission the temperature was "jumpy." Never above 100, it was often about 99, though at other times it was subnormal. The temperature was irregular. It is this feature, taken in conjunction with the memory of a previous mistake which I made in missing an endocarditis of this kind, and attributing the condition to dilatation, which makes me offer this *caveat* here. We do not anticipate any particular clinical changes in this case. It is probable that he will gradually improve; it is even possible that he will eventually recover, but this will take a long time. In the meantime rigid rest is the treatment. Digitalis will not have any demonstrable effect. Altogether, one might say, a rather uninteresting case. But it illustrates essentially the curious apparent discordance between subjective symptoms and the gravity of the real state of affairs met with in cases of dilatation.

No one can deny the seriousness of dilatation; in adults it is, next to alternation, the most serious of conditions. But time and again one has found a striking lack of subjective distress. Two illustrations bearing on this point will suffice, I think, to emphasise this. A patient, a big, well-developed man, came into hospital for dropsy of the legs. The pulse was always very quick, dulness increased. Other signs of passive congestion present. Persistently, however, this man denied any feeling of ill-health, and took his food with refreshing zest. He

left hospital at his own request, but in a few weeks was dead. A young girl with the same general symptoms presented exactly the same apparent subjective well-being. She, too, in a few months died. It is familiar, too, that with the onset of dilatation, perhaps a terminal event in a case which had had for years various subjective signs, these pass away, œdema supervenes, and death closes the scene.

CASE 4.—*Rheumatic myocarditis. Œdema. Angina. Death.*

E. M—, a well-nourished boy, æt. 10 years, had a first attack of rheumatic fever in the spring of the present year. He never recovered completely and has been weak and exhausted ever since. The weakness had progressed and, when seen by the writer on May 21st, he was obviously very ill indeed. He was admitted the same day into hospital. The pulse was 120 and regular.

Condition.—The boy was pale, not wasted. He was breathing rapidly, but not in a laboured fashion. There was œdema of the legs. There was not much venous pulsation in the neck, but the epigastrium heaved forward with each beat of the heart. The heart was generally enlarged as noted by percussion and by the X rays. A loud systolic murmur replaced the first sound at the apex and was conducted to the back. It was heard at all areas over the præcordia. The respiration was markedly accelerated, the movements were shallow, and at the bases moist *râles* were to be heard.

The liver was enlarged and tender. No albumin in the urine. The clinical picture was one of advanced cardiac muscle failure. To say this, however, is not sufficient; at least, some indication of the cause must be offered. The condition was, we thought, one of two things. Either there was loss of tone in the muscle with consequent dilatation, loss, that is to say, of a definite property of the myocardium; or—and this it proved to be—there was loss of the power of contractility, again, a definite myocardial property. Conceivably both conditions were present. Of these two conditions, dilatation was the more probable. Recognised by the extension of the area of cardiac dulness especially to the right, a sustained high pulse-rate, and the general signs of œdema, it is a condition not uncommon, especially in youth, and with prolonged rest may be completely recovered from, though less often does this happen in adults. Exhaustion of contractility, on the other hand, may or may not be associated with evidence of dilatation, the pulse may not be accelerated, but, and this is the diagnostic and essential element, there is to be detectable, by suitable means, the pulsus alternans, a rhythm in which large and small beats regularly alternate, the rhythm remaining regular. This condition is met with, *par excellence*, in the failing heart of chronic interstitial nephritis. Its significance is extremely grave, and, unlike cases of dilatation in the young, the conditions inducing this failure of contractility are almost invariably fatal. Frankly, however, it was not this condition which we expected to find in this case. At such an early age it is distinctly uncommon. It happened, nevertheless, that in the routine electrocardiographic examination carried out with the patient resting in bed, the electrodes from the patient leading into the galvanometer room elsewhere, that the resulting curve clearly showed a brief phase of alternation. This was distinctly surprising, and I thought that, perhaps, as the respiratory rate was exactly half of the pulse-rate, the appearance might be due to respiratory interference, a pulsus paradoxus, with the smaller beat coinciding with the inspiratory phase. I consequently rigged up in the galvanometer room appliances which enabled me to gain simultaneous photographic curves of the radial pulse, the respiratory movements, and the electric curve. The result, however, showed that the true condition was, after all, alternation. The prognosis immediately assumed a graver hue. Up to this time he had had no pain. On May 24th, he had a sudden attack of true and severe angina pectoris. (He was said to have had a similar attack a fortnight previously.) The pain was very severe and localised especially in the vicinity of the left shoulder. The face on this side became distinctly puffy soon after. Amyl nitrite afforded relief. Similar attacks occurred on June 3rd, 8–9th, 23rd, 26th, and 30th, each paroxysm leaving the patient weaker than before. The œdema increased, albumin appeared in the urine for the first time on May 24th, persisted till June 3rd, then disappeared, to reoccur on June 11th, since when it has persisted. The quantity of urine passed was scanty. Cough, nausea, drowsiness, and progressive exhaustion added increasing distress. Digitalis at one stage, June 22nd to July 5th, was vigorously pressed.

The only result was to induce some nausea and to cause the pulse, which had throughout been rapid, to become slow and irregular. Electric curves taken at this time showed the arrhythmia to be due to very marked sinal depression and the production of markedly anomalous ventricular complexes, suggestive of unequal and varying blocking effects upon the AV bundle and its branches. The boy ultimately died, and a necropsy showed a large heart, with definite stenosis of the mitral valve.

Comments.—There are three points of interest. One is the evidence of the extraordinary intensity of the poisoning of the heart from the rheumatism. Whether the valves were affected or not is a matter of relative indifference; it was the myocardium and its most vital properties which were irreparably damaged. The second point is the clinical identity of the picture with that seen in adults. So definite a picture, the œdema, the angina, the pulsus alternans, is in the nature of a syndrome and presents in a lad, æt. 10 years, all the detail as definitely as in the broken-down man of 60. The third point is the record of pulsus alternans and angina at so unusually early an age as 10.

December 13th, 1915.

Congenital Tuberculosis.—Péhu and Chalier have collected from literature 16 undoubted cases which have been published since or were not included in their last paper on this subject. In 12 there were naked-eye lesions, and in 4 there were no microscopic lesions, but tubercle bacilli were present or animal inoculation was positive. They conclude that authentic cases of congenital tuberculosis are very rare. There are only 51 on record. The conditions under which they occur are always the same. They appear during or at the end of pregnancy when the placental circulation is established, and result from a bacillæmia which is usually a terminal event. They should therefore be regarded as examples of transplacental contagion, and not of direct heredity.—*Brit. Journ. of Children's Diseases*, p. 339.

RUPTURED TUBAL GESTATION, RESULTING IN HÆMORRHAGE INTO THE GENERAL PERITONEAL CAVITY.

By H. T. HICKS, F.R.C.S.

In spite of the desperate condition of the patients, ruptured tubal gestation must be classed as one of the most satisfactory surgical emergencies within the female abdomen. Out of 26 cases operated upon, one was fatal. In this there was an interstitial gestation, and I was forced to remove the uterus. The operation added much to the already shocked condition of the patient. There are a few points in dealing with these cases which are worth special consideration.

The Difficulties in Diagnosis.

In most cases there will be little or no difficulty. The blanched condition of the patient will at once suggest internal hæmorrhage, and if there is a history of a missed period, of acute pain in the lower abdomen, followed by vomiting and a little later by a blood-stained vaginal discharge, there will be no difficulty in confirming the symptoms by the physical signs. In order to understand exceptions, one must keep in mind the pathological anatomy of tubal gestation.

The Fallopian tube is divided anatomically into three parts: (*a*) interstitial; (*b*) isthmial; (*c*) ampullary portions.

An ectopic ovum may be held up in any part of the tube, and the symptoms will vary accordingly. I will leave interstitial gestation alone, because it is less common than the other varieties, and, since the ovum is in fact placed within the muscular wall of the uterus, the course which the pregnancy may run is obviously much more complicated than in isthmial or ampullary forms. The one great difference between isthmial and ampullary gestation is the time of the onset of the symptoms. In isthmial gestation the trophoblasts of the ovum may erode the thin walls of the tube as early as the tenth day and seldom later than the third week. As soon as the blood begins to escape into the peritoneal cavity there will be a sudden attack of pain, accompanied by vomiting. In a few hours

the patient begins to show signs of internal hæmorrhage. The ovum is not much larger than a pea or small bean, and the hæmorrhage is not violent. There is merely a small leak going on, and although the patient may at first show signs of collapse, this is due rather to shock from the severe pain than to actual hæmorrhage. There is generally plenty of time in these cases. In isthmial gestation the hæmorrhagic discharge from the uterus is slight or absent. In fact, at the onset it is the exception rather than the rule. The uterine hæmorrhage which is so characteristic of later ruptures is due to the casting off of the intra-uterine decidua, which occurs when the ectopic ovum's life is either seriously threatened or it is dead.

Thus in most cases of ruptured ectopic gestation at the isthmus of the tube, there is usually one attack of pain, followed by collapse, and the blood which is slowly poured out seldom becomes encysted with the formation of a hæmatocele.

If one contrasts this picture with that which occurs if the ovum is situated in the ampulla of the tube, there is a marked difference.

In ampullary gestation the ovum is older, and there may be 1, 2, or even 3 missed periods. The fate of the ovum is much more varied, and on account of the more advanced development of the placenta the blood is poured out into the general abdominal cavity at a much greater rate. When once there are signs of hæmorrhage into the general peritoneal cavity, and one or more periods have been missed, the gestation is in the ampulla of the tube, and there is no time to lose. When the tube attempts to expel the ovum (tubal abortion) or rupture takes place at the ampullary end the blood at first collects around the fimbriated end of the tube and is in close relation with the ovary and the peritoneum covering the posterior aspects of the broad ligament and sides of the pelvis. It happens, therefore, that the peritoneum at once reacts and makes an attempt to encyst the blood. This it may succeed in doing, and a hæmatocele is formed of varying size. This is the most common end to an ampullary gestation and may be termed an attempted natural cure. In many cases, however, the ovum does not die and the placenta still erodes. The result is that further bleeding takes place. This process may occur even 3 or 4 times, until the blood breaks through and a general intra-peritoneal bleeding takes place. The symptoms of a rupturing ampullary gestation differ from isthmial ruptures in the following manner:

(*a*) There is never a single attack of pain and collapse. Each bout of bleeding gives rise to pain and vomiting, followed by more or less collapse, until the final rupture takes place followed by great collapse.

(*b*) Hæmorrhage from the uterus is an almost constant symptom, because the ovum's life is seriously threatened several times, and the intra-uterine decidua begins to disintegrate soon after the first threatening. The decidual cast will be thrown off when the ovum is dead. The diagnosis in ampullary gestations is, as a rule, a more simple matter, because all the classical signs of ectopic gestation are present. It is to be remembered that the attacks of pain may be repeated several times. In the early stages the condition may be mistaken for a threatened abortion associated with a small pelvic tumour. The symptoms are akin, but any doubt may be cleared up by the presence of great tenderness on examination and by the character of the pain. The pain caused by threatening rupture of a tubal gestation is most acute and severe, while in simple abortion the pain is merely that of uterine colic. There are, of course, other differences, such as the size of the uterus and the condition of the cervix, which are too simple to mention. Later on there can be little difficulty in making a diagnosis. The history, the acute tenderness, the blood-stained discharge from the uterus, and the collapsed condition of the patient will at once tell the tale.

I think there is often much greater difficulty in coming to a decision when an isthmial gestation has ruptured. The most common condition which may cause trouble in this direction are perforating gastric ulcer, acute appendix, and torsion of a small ovarian tumour.

The general condition of the patient and the history may throw the necessary light upon the case, but sometimes one has to rely completely on the examination of the patient. The site of the acute pain in a ruptured ectopic gestation is usually in the lower part of the abdomen, but quite a number of these cases complain of intense pain in the epigastrium. This may lead one to suspect a perforation of stomach. It will be found that if the pain is severe in the epigastrium the patient had probably been lying down at the time of

rupture. Thus the blood very quickly flows upwards into the epigastric area. Another point to remember is that of all the fluids that collect in the peritoneum, there is none that causes so much tenderness on pressure as blood.

I have frequently settled a doubtful diagnosis by making a vaginal examination and noting the extreme tenderness in Douglas' pouch. Free blood in the peritoneum does not cause much rigidity, and to detect free blood by percussion needs great care. The intestines distend and float on the blood. When the position of the patient is changed the dependent part does not fill immediately, so that a little time should be given for the blood to gravitate downwards.

Operation.

The one great point about the operation is rapidity of action. No time is to be lost, and rarely need the operation take more than fifteen minutes.

As soon as the peritoneum is opened the dark coloured blood will flood the wound. It is useless to sponge this up, for more will rise up, and much valuable time will be lost. The operator plunges his hand down into the pelvis and locates the enlarged uterus. He will already have some indication as to whether the ovum is in the isthmus or in the ampulla. If the former, he will run his fingers along the tubes, and will come on the small bean-like ovum. This is pulled up through the blood, ligatured, and the abdomen closed after sponging out as much blood as possible. If the ovum is at the ampulla, the pregnant tube will be found forming a mass with much blood-clot in its immediate vicinity. What adhesions there may be are mostly recent and will cause little or no trouble.

It has often been stated that old salpingitis is a common cause of tubal gestation. This is no doubt true, but if an ectopic gestation follows on a past attack of salpingitis, the ovum will always be found lying in the tube, which has either recovered or escaped the inflammatory attack. In 3 cases I have found this was so most certainly, and in 1 case I lost a lot of valuable time in attempting to bring up a very adherent mass on the left side, which consisted of ovary and tube and was the remains of a recent pyosalpinx. The patient was very collapsed, and I was in two minds as to whether we could save her. I turned to the other side and found the right tube quite free from adhesions, the fimbriated ends were open, and the ruptured sac of the ovum was lying about 2 inches from the uterine end of the tube. About 3 weeks ago I had just exactly such another case. When I passed my hand down I felt a fixed mass about the size of a small orange lying to the left of the uterus. I tested its mobility and found the adhesions were old and tough. I at once turned to the other side, and with no difficulty brought out a tubal mole and completed the whole operation in less than 15 minutes. This patient was almost pulseless when she was admitted, but made a rapid recovery. I wish to bring out this practical point, for I am convinced that undue delay during the operation is almost fatal. These patients will not stand much more than 20 minutes, and the more rapid the operation the less will be the mortality.

I have frequently heard it said that when these desperate cases are in a condition of extreme collapse, it is advisable to get them warm and stimulate the circulation before operating.

If a patient falls down in the street, and the history points to isthmial gestation, there may be a certain amount of shock apart from that of the internal hæmorrhage. In these cases the bleeding is slow, and the onset is frequently followed by some recovery of the pulse. To apply the same idea to an ampullary gestation would be most unwise. Here the ovum is older and the bleeding more severe. It is much safer to operate on all cases without delay. There should be no waiting for recovery of the pulse. During the last year 3 cases have died on their way to the hospital.

Sometimes these patients look worse than they really are, and I have yet to find one who will not stand a very rapid operation, followed by infusion of saline fluid whether poured into peritoneal cavity, given *viâ* a vein or otherwise. This is desperate surgery, but it is well repaid.

December 13th, 1915.

Round Worm in a Tonsil.—A. B. Middleton reports the case of a girl, æt. 8 years, who had not been well since an attack of chicken-pox at the age of 2. She was anæmic, thin, and undersized, and had an adenoidal expression, but had never suffered from sore throat. She had had regular bilious attacks for 2 or 3 days once every three weeks. Both tonsils were enlarged. Their enucleation and removal of adenoids were undertaken. The right tonsil ruptured when the snare was tightened, and a female ascaris, 1¼ in. in length, escaped. Within the tonsil was a cavity, possibly the habitat of the worm for several years. After the operation the bilious attacks ceased and the child improved in health.—*Brit. Journ. of Children's Diseases*, p. 348.

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*Specially reported for the Clinical Journal. Revised by the Author.

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We regret that owing to the war the number of pages must be diminished for a time.

ON TOXIC POLYNEURITIS OF THE MOTOR TYPE.

By J. MICHELL CLARKE, M.A., M.D., LL.D., F.R.C.P., Lt.-Col. R.A.M.C.T.,
Joint Professor of Medicine, University of Bristol, and Physician to Bristol General Hospital.

IN the following cases of multiple neuritis objective disturbances of sensation were absent, and though there was a certain amount of pain, in one case considerable pain, the symptoms were essentially those of motor paralysis. Instances of this type of neuritis, excluding, of course, those of single nerves, are not at all common. The great majority of cases of multiple neuritis are alcoholic, and sensory disturbances form one of the most striking clinical features. In the early stages of the motor type of polyneuritis, when the disease is spreading and invading successive groups of muscles, the difficulty of diagnosis from an ascending spinal paralysis, and also from certain cases of anterior poliomyelitis may be difficult. Moreover, many of the most characteristic features of multiple neuritis are absent or inconspicuous. Thus in alcoholic neuritis women are far more often affected than men, and women especially between 40 and 50. The legs are involved first, and in the majority the arms escape or are comparatively slightly affected, whilst pains, tenderness of nerves and muscles, and peripheral loss of cutaneous sensibility are prominent, and these symptoms, with certain other features, especially the mental changes, which form the well-known Korsakoff's syndrome, and the cardiac weakness, due to the action of alcohol on the heart-muscle, all contribute to make up a characteristic and striking clinical picture. Again, in acute toxic polyneuritis the cause is generally obscure. Except in those cases which follow influenza, or some such infective disease, the onset is insidious, may take place during apparent good

health, and the absence of constitutional disturbance seems to negative the idea of infection.

During the first few days of a case of toxic polyneuritis of the motor type the resemblance to ascending or Landry's paralysis is close, so that one may have to postpone the diagnosis, and may have considerable anxiety, owing to the far greater gravity of the prognosis in the latter disease. In making the distinction between the two affections one must bear in mind that it is a clinical one, and that in the light of recent pathological investigation into some cases presenting the typical features of Landry's paralysis, it is safer to use this name entirely in a clinical sense, for there is no doubt that a certain proportion of cases of Landry's paralysis are due to neuritis. Pfeiffer* has recently published a case of this kind fatal on the 7th day, in which interstitial neuritis affecting the nerve-roots and peripheral nerves was found, yet no sensory symptoms occurred. In his paper references are given to other cases of the kind. With this reservation it can be stated that in the majority of cases which conform clinically to the type of polyneuritis the onset and progress of the paralysis is slower than in a typical case of Landry's paralysis, and in the course of the second or third week, if not at the onset, the paralysis begins to be definitely greater at the periphery of the limbs, if not in all four extremities at first, yet either in the arms or in the legs, and to predominate in the extensors, whilst alterations in the normal electrical reactions of the most affected muscles begin to be evident. These changes in electrical reaction precede the wasting, which, indeed, in some cases is not very marked throughout the illness.

The diagnosis from acute anterior poliomyelitis is not so difficult, as in the great majority the sufferers from this disease are much younger than those from acute toxic polyneuritis, and if in exceptional cases the age cannot be taken into account in distinguishing between the two affections, there are the decided signs of constitutional disturbance at the onset of acute poliomyelitis, the characteristic pains on passive movements of the joints, the absence of such symmetrical paralysis, the escape of a muscle or group of muscles in the otherwise paralysed limb, the recovery of some muscles soon after the onset, and the frequent presence of a large lymphocytosis in the cerebro-spinal fluid.

The first case I report is of the senile type, and corresponds to the usual descriptions in its insidious course, and absence of pain or other sensory disturbance; but although sensory symptoms are usually comparatively inconspicuous, they are rarely so completely absent as in this instance, in which also there was not the marked arteriosclerosis which is generally associated with senile neuritis. The diagnosis presented no difficulty, as the signs of polyneuritis were characteristic by the time the patient came under observation.

This patient was an emaciated man, æt. 72 years. The family history was good. He had never had any illness, had always been strong, healthy and temperate, and worked hard as a blacksmith. Six weeks previously to admission he felt quite well on getting up, but on going to work noticed that his right leg dragged; he was, however, able to stay at work without inconvenience during the whole of that day, but on going to bed he had to be helped upstairs, as both legs were so weak that he had great difficulty in getting upstairs even with help. He had a good night, but on waking the next morning he found the weakness had so increased in the legs that he could not stand, and had spread to both arms, so that he could not raise himself in bed. There was no constitutional disturbance of any kind and no pain. The limbs felt "dead" and numb. Since this time he has remained *in statu quo*, confined to bed and practically helpless. Sphincters, speech, swallowing, and special senses unaffected.

On admission he was very helpless and unable to move himself in bed. Tongue clean. Pulse 104. Respiration 24. Temperature subnormal. The few teeth remaining were bad. No blue line on gums. There was moderate atheroma of the vessels. Both legs were powerless except for slight movements at left hip. The feet were dropped, and toes flexed. There was also double wrist-drop, the fingers were slightly flexed and could not be extended. With great effort the right arm could be moved feebly at the shoulder, and very weak movements of the left were possible at shoulder and elbow, with feeble flexion of wrist. He could not feed himself nor use the hands for any purpose.

* "A Case of Landry's Paralysis with a special reference to the Anatomical Changes," J. A. J. Pfeiffer, 'Brain,' vol. xxxv, pt. iv, p. 293.

The head and neck could be moved freely. The muscles of all limbs were wasted, especially the extensors of wrist, fingers, ankles, and toes.

Diaphragm and intercostal muscles acted well.

Electrical reactions. Loss of reaction to Faradic current in extensors of left wrist and fingers, interossii of right hand, anterior tibials and peronei muscles of both legs; very feeble reaction to strong current in long extensors of right wrist and fingers and muscles of thumbs. The flexor muscles responded but feebly in arms; calf muscles also reacted feebly. All muscles reacted to the constant current, but the extensors gave a slow wave, and ACC = KCC. In the flexors the response was normal.

There was no affection of sensation to pain, light touch, heat or cold. Sense of position and of movement of limbs unaffected. No muscular tenderness. There was a slight tremor of the left arm when movement was attempted. No cramps or spasms.

Reflexes.—Deep reflexes absent. Superficial: abdominal and cremasteric present, plantar absent.

The paralysis was entirely flaccid in type. Cranial nerves unaffected. Pupils equal, acted well to light and accommodation. Optic discs normal. Thoracic and abdominal organs normal. Sphincters unaffected.

He remained in the hospital one week, his condition being unchanged, and then refused to stay.

In the next case the diagnosis was for some little time doubtful, owing to the completeness of the paraplegia, and to the extensors not being predominantly affected until late. For a time also the electrical reactions were unaltered. Wasting of the muscles did not occur for a long time, and was never conspicuous throughout the illness. In fact, the slightness of the wasting in muscles which remained paralysed for months was remarkable. The pains in the cervical spine soon after the onset also introduced a doubtful element in diagnosis. The time taken for recovery, a little over 12 months, shows what is to be expected in a severe case of this type, and also that even if little progress is made for 6 months this need not preclude complete recovery. The case, indeed, illustrates that recovery may be perfect, if due care is taken throughout the illness in the prevention of contractures and of unfavourable position of the limbs. So favourable a result is hardly seen in any other form of paralysis. Throughout the illness there was no affection of sensation, beyond at one time a doubtful and partial anæsthesia to light touch on outer sides of the feet. Efforts were made to find out the nature and source of the toxin, but without success. Examination of the blood and cerebro-spinal fluid and bacteriological examinations of the fauces and urine gave no clue to it.

A joiner, æt. 34 years, had always enjoyed good health, except for a mild attack of jaundice when in the Argentine 5 years previously. He was in a very healthy district, and never had malaria. He had never had syphilis, and had always been temperate. Eight weeks previously he had a sebaceous cyst removed, which left a discharging sore for 3 weeks and then healed well. He had no sore throat. On September 14th, 1913, he had cramp in the muscles of the right leg, followed next day by stiffness in both legs, then by weakness. The stiffness and weakness gradually increased until the 18th, when his feet tingled and became so numb that he could not tell their position, and consequently found great difficulty in walking. On the 19th he could not walk to his work, but managed to get there on his cycle. On the 20th he was unable to walk without support, as he lost his balance, and was unable to tell the position of his legs unless he looked at them; numbness and tingling, with weakness, also appeared in both hands. On the 23rd he felt a feeling like a "lump of lead" in the abdomen. From this date the weakness of the limbs steadily increased, and he was confined to bed until his admission to hospital on October 2nd.

On admission he was well nourished and healthy looking. The pulse was 88, the respiration, tongue, and temperature normal. The thoracic, abdominal organs, and urine were healthy. He was unable to stand, but could just move the legs slightly at knee and hip-joints, otherwise they were paralysed. The paralysis was flaccid. There was no foot-drop. The muscles were not wasted—they all reacted to a Faradic current. The constant current gave so much pain that it could not be used. There was no affection of sensation to pin, cotton-wool, heat or cold. The sense of movement and of position was not lost.

Reflexes.—The deep reflexes in the legs were absent, in the arms absent or very feeble.

Superficial reflexes.—Plantar absent, cremasteric present, abdominal very feeble. No pain on passive

movement of limbs. Slight loss of power in forearm and hands. Special senses and cranial nerves unaffected. No optic neuritis; no alteration in speech or swallowing. Sphincters unaffected. Spinal column normal; no tenderness over spines. Cerebro-spinal fluid on lumbar puncture gave a slight excess of cells, lymphocytes and endothelial cells. Wassermann's test negative for blood and cerebro-spinal fluid. A swab from the throat showed no Klebs-Loeffler or diphtheroid bacilli. Blood normal: no leucocytosis. Bacteriological examination of urine negative. Stools appeared healthy. It was impossible to diagnose the exact nature of the lesion causing the slowly spreading paralysis of the legs. The mental condition was clear and good.

During the next two weeks he suffered much pain in the neck and back of the head. At the end of this time paralysis of both lower extremities had become almost complete, except for slight movement of the hip-joints; there was now marked foot-drop on both sides. Numbness (without objective loss of sensation) and loss of power in the hands had increased, each grasp registering only 15 on the inner dynamometer scale, and the deep reflexes in the arms were now completely abolished. There was still no muscular wasting, and his condition was otherwise unchanged. A few days later the abdominal reflexes disappeared, and he complained much of girdle pain. Three weeks after admission electrical reactions showed in the legs diminished reaction to interrupted current, especially in dorsi-flexors of feet and long extensors of toes. To constant current the calf muscles gave normal reaction. The quadriceps extensors reacted only to a very strong current: Right KCC = 10 m.a.; Left KCC = 14 m.a. ACC = 18 m.a. Peronei and tibialis antici on both sides KCC = 1 m.a. ACC = 2·5–3 m.a., and the contraction wave was slow.

On November 6th there were slight signs of wasting in the leg muscles, especially marked in the anterior muscles of the legs below the knee. The muscles were now also distinctly tender to pressure, and there was pain on passive movements of the ankles. There was a definite reaction of degeneration (R.D.) in peronei and tibialis antici muscles, and also some loss of sensation of movement and of position in left leg. Sensation otherwise unaffected.

By the end of November the paralysis had increased in the forearms and hands, which had become very weak, and the muscles of the forearms and the interossei appeared to be wasting. He could feed himself, but not cut up his food. There was indefinite and slight loss of tactile sensation on outer borders of feet. The only movement in the lower extremities was internal rotation at hip-joints. The muscles of the lower extremities gave a slow, very feeble response to a very strong Faradic current; those of the forearm, especially extensors, and of the hand also reacted badly, whilst the arm muscles gave a normal response. The nerves responded well. The leg muscles (below knee) now all showed reaction of degeneration to constant current. He remained much the same until the middle of January, when the forearms and hands began very slowly to regain power. The small muscles of the hands had wasted, and showed occasional fibrillary twitchings. During this time all the muscles had become tender to pressure. The foot-drop had increased, and the legs were now placed in splints. Sensation remained normal and the bladder unaffected. There was continued constipation. He could not sit up in bed. The diaphragm was never involved. All the reflexes in the limbs were lost, but the cremasteric and abdominal reflexes, though feeble, were present. The only muscles of the legs that responded now to the Faradic current were the right gastrocnemius and tibialis anticus, and adductors of left thigh.

During March he slowly improved, regaining first power in arms and hands, and then in hips. The muscles were no longer tender. In April he could sit up in a chair, swing his legs backwards and forwards, and slightly extend the left leg. There was, however, yet no recovery of power in muscles below the knee. In May he could very nearly stand without support, could walk with the aid of a walking machine, and by the end of the month could walk by pushing a chair in front of him. The foot-drop still remained for a long time afterwards. The subsequent course has been very slow but steady recovery, so that he is now able to get about, and is going to take up some light employment. The knee-jerks are still absent.

In the following case, as in the preceding, the cause of the disease could not be ascertained, the onset was insidious, whilst the patient was apparently in perfect health. His occupation exposed him to extremes of heat and cold, and often involved heavy muscular work.

The distribution in this case is peculiar, and differs from the two just reported. The disease began in the upper extremities, with two days later weakness in legs, not proceeding to actual paralysis for 3 to 4 days longer. Moreover, in the legs the thigh muscles were chiefly affected, and those below the knee at first escaped. The paralysis in all four extremities was, however, strictly symmetrical, and from the onset was in the arms of the peripheral type, extensor paralysis predominating, and the extensor muscles alone showed commencing reaction of degeneration (R.D.). In the lower extremities the quadriceps extensors were especially affected, and showed more wasting and alteration of electrical reactions than any other muscles of the body. In spite of the somewhat unusual distribution of the muscular paralysis there is no doubt of the case being one of toxic polyneuritis. As in the other cases there is a complete absence of sensory affection, subjective or objective, apart from pains in the right shoulder at the onset. This was the only fatal case.

A marine fireman, æt. 50 years, had never had any illness and denied venereal disease. He had six healthy children. He had been moderate as to alcohol, but smoked about one ounce of tobacco daily. Three weeks before admission, whilst in perfectly good health, he had pains in right shoulder. On the following day he went to work, but felt weakness in both arms and hands; on the third day the weakness had so increased in the arms that he was unable to work. He felt some weakness of the lower extremities, which did not increase, and he was able to get up and sit about until 3 days later, when, on getting out of bed, his legs gave way and he fell. He remained in bed practically helpless for 10 days, and was then admitted. There was no pain after the first day; some tingling and feelings of pins and needles in forearms, hands, legs, and feet. He had had some headache, the mind has been quite clear. Profuse sweats occurred at night, but no fever. Sphincters unaffected. He did not come into contact with lead in his work.

On admission he was well-built, healthy-looking, and well nourished. Gums and teeth healthy. He lay in bed on his back, with arms across the abdomen. Except for some movements in the shoulder, chiefly due to pectorales, serratus magnus, and trapezius muscles, the upper extremities were completely paralysed. When they were raised there was double wrist-drop. He could not sit up, turn, or move in bed. In the lower extremities slight rotation of hips and slight dragging up of legs by thigh muscles were possible, the ham-strings and extensors of legs were paralysed, but he could flex and extend ankles and toes freely but feebly, with this exception the lower extremities showed complete paralysis, could not be raised off the bed, and were useless.

The paralysis was of the flaccid type. Slight wasting of muscles was noted in the left quadriceps extensor, perhaps also in the right, in the extensors of wrists and fingers, and in the muscles of the thumbs and the abductor indicis. It was nowhere conspicuous, and the other muscles preserved their normal size.

There was no paralysis of cranial nerves. Pupils equal, dilated, reacted to light and accommodation. Optic discs normal. Speech and swallowing normal. Movements of head and neck unaffected. The chest moved well in respiration, the movements of the diaphragm were perhaps a little feeble. Sensation to touch, pin-prick, heat and cold everywhere normal. He retained the sense of position of his limbs; passive movements were not painful, and the muscles nowhere tender.

Reflexes.—Deep; biceps and triceps jerks present on both sides, wrist, knee, and ankle-jerks absent.

Superficial.—Abdominal absent, cremasteric present, both plantar present and flexor in type.

Bladder unaffected; bowels constipated. Pulse 80, regular. Respiration 24. Thoracic and abdominal organs normal. Urine 1026, no albumin, no sugar, loaded with urates. Cerebro-spinal fluid under normal pressure and clear. Cells normal, only a few lymphocytes being present. Wassermann's test negative.

Examination of blood.—Red cells 6,000,000 per c.mm., hæmoglobin 90 per cent., leucocytes 19,000 per cm., polymorphs 69 per cent., mononuclears 30 per cent., eosinophils 1 per cent.

One week after admission electrical reactions were as follows:

Interrupted current.—Normal response in all muscles except the extensors of wrists and fingers and quadriceps extensor on each side, where a strong current was necessary, and to a less degree in small muscles of hands.

Constant current.—Right arm, in long flexors and extensors, KCC = ACC, 7 and 5 ma. respectively.

Left arm, long extensors, wrists and fingers, KCC = 8 ma., ACC = 6 ma. Flexors, wrists, and fingers, KCC = 5 ma., 7 ACC = 8 ma. Right abducto indicis, KCC = 3 ma., ACC = 7 ma. Left KCC = 6 ma., ACC = 7 ma. Legs: right quadriceps extensor, KCC = 12 ma., ACC = 10 ma.; left quadriceps extensor, KCC = 18 ma., ACC = 12 ma. Other muscles gave normal reactions.

Ten days after admission the paralysis of the arms was unchanged, wasting of the forearm muscles was more evident; the diaphragm was slightly affected; there appeared to be little if any power in the abdominal muscles, to which the constipation was partly attributable; the thigh muscles were slightly more wasted; and the paralysis had slowly spread to the legs, so that hardly any movement of feet and toes was possible, and the right foot was dropped. Plantar reflexes very feeble. Other reflexes absent. Sensation good everywhere.

During the next few days the action of the diaphragm became increasingly feeble, the respiration was quickened, the pulse became increasingly rapid and weak, the heart's action feeble, and he had difficulty in swallowing. Movements of the tongue and speech were unaffected, the facial and oculomotor nerves were not involved. He died of heart failure two weeks after admission. A necropsy was not permitted.

A woman, æt. 33 years, had been married about 18 months. Before marriage she was completely worn out by nursing her mother through a long illness. Her father died of diabetes. She had had no previous illness, and had always been temperate. The illness began with pains and loss of power in the legs 2 or 3 months before her confinement, which took place about 3 months before admission. For 2 months previous to confinement she lay in bed with her legs drawn up, and screamed if they were extended. Delivery was normal. After it the paralysis spread to the arms. The pains in the legs began some time before she lost power in them. There had been no pain in the arms, but they felt dead or numb. The limbs rapidly wasted, and for 2 months before admission she had been helpless and unable to feed herself. From the fifth month of pregnancy and continuously since she had suffered from salivation.

On admission she was emaciated, and the muscles everywhere were extremely wasted. She lay on her left side with her knees fully flexed, and thighs flexed on abdomen. The legs were contracted in that position, and she screamed when they were moved. Appetite good. Tongue furred and tremulous. Pulse 120, respiration 20. Temperature normal. Bowels constipated. The teeth were very bad, many carious stumps, gums spongy, with pus around roots of teeth. No blue line on gums, no evidence of any source of lead poisoning. The salivary glands were not enlarged: there was an almost constant flow of saliva. The thoracic and abdominal organs appeared to be normal. Urine normal. The pupils were unequal, but reacted well to light and accommodation. Optic discs normal. There was paresis of muscles of left side of face. Swallowing and speech were unaffected. She complained of some pain in the neck, but movements of head and neck were free.

Arms.—She could raise the arms, and flex and extend the elbows partially. All the muscles of arms and forearms were much wasted. There was double wrist-drop, the small muscles of the hands were wasted, the thumbs and index fingers were in position of extension, the other fingers flexed. The left index finger could be flexed, and the thumbs could be partially opposed and adducted; otherwise paralysis was complete.

Trunk.—The abdominal muscles and those of the back were much wasted; she could not sit up.

Legs.—Glutei were wasted, and there was great wasting of all the muscles of the legs, especially below the knee, where the anterior tibial and peronei had practically gone, leaving a hollow. The ham-strings and calf muscles were relatively well preserved. There was complete double foot-drop, and all the toes were in position of flexion.

The only movements possible were feeble flexion of the hips, and slight movement of the legs. From contraction of tendons and adhesions in the joints neither hips nor knees could be passively moved beyond a right angle; and such movements caused much pain. None of the muscles were tender, and there was no tenderness over the course of the nerves.

Electrical reactions.—To Faradism muscles of arms acted well; those of the forearm, especially extensors, feebly. Small muscles of hands reacted well; the quadriceps extensor and ham-string muscles gave a weak reaction. The calf muscles contracted to a strong current. No reaction was

obtained from the peroneal nerves, peronei, tibialis anticus and long extensors of toes. The constant current gave her great pain, and the reactions could not be well taken, but no reaction of degeneration was found save in the muscles of the anterior and outer aspect of the legs.

The deep reflexes were absent, abdominal present, plantar absent. There was nowhere any affection of sensation to light touch, deep pressure, pin-prick, heat, or cold. The sense of localisation of touch and of position of limbs was not lost. The sphincters were unaffected. Her mental condition was childish, she cried easily; but there was no loss of memory or confusion of ideas.

The first point in treatment was to get the limbs into better positions. She was therefore anæsthetised, numerous firm adhesions in and about the hip, knee, and ankle-joints were broken down and the legs placed on straight splints. The upper extremities were also freely moved at all joints. Subsequent treatment was by gentle massage and passive movements and Faradism, with maintenance of the limbs in suitable postures in order to prevent dragging on the paralysed muscles. The salivation gradually ceased. After 5 months she had recovered sufficiently to return home.

This is an example of the somewhat rare form of multiple neuritis which occurs in pregnancy, or during the puerperium. If the onset is during pregnancy, the symptoms may become marked after labour, which, as in this case, may be normal. The disease appears especially in patients who suffer severely from the vomiting of pregnancy. In puerperal cases the cause in generally held to be a septic infection; in those arising during pregnancy it is attributed to an intoxication related to the physiological processes of that condition, or by others, *e.g.* (Huber) to infection from a previous puerperium or from uterine disease (Oppenheim). In some cases the progress of paralysis is rapid, and the disease may take the form of Landry's paralysis. The possibility of alcohol as an additional factor must be remembered. In this case there was no evidence of alcohol, but the patient was exhausted by long nursing before she became pregnant. The prognosis in this form of neuritis is as a rule favourable.

In the last case the chief symptom was inco-ordination, combined later with paralysis. Muscular weakness is important in the diagnosis from tabes dorsalis, because it is not a feature of this disease until, at any rate, a late stage. The ataxy in this was as extreme as in a bad case of tabes dorsalis. Moreover, it was attended by marked hypotonus, so that the joints and limbs could be placed in very abnormal positions without causing discomfort. The loss of the muscular sense and of that of position of limbs was also pronounced. Some doubt has been thrown on the existence of severe ataxia in multiple neuritis, it being held that such cases are really examples of tabes dorsalis, in which the association of disease of the peripheral nerves with that of the posterior columns of the cord, which frequently occurs, is more pronounced and extensive than usual. Judson Bury* says (1) that ataxia is rare in peripheral neuritis even when cutaneous and muscular sensibility are profoundly affected; (2) that it occurs occasionally when signs of muscular weakness and of diminished cutaneous sensibility are slight or absent. Undoubtedly marked ataxia is rare in multiple neuritis, especially when, as in this case, there is no affection of common sensation. In the diagnosis in this case I would lay stress on the complete absence of all signs of affection of the spinal cord, and on the presence of a definite amount of paralysis in the legs of the peripheral type, symmetrical, and accompanied by some muscular wasting, and on recovery in a comparatively short time. Another point is the affection of the facial nerve, and its recovery after a short time. This cranial nerve is the one most commonly affected in multiple neuritis.

Another feature was the presence in the wasted muscles of fibrillary tremors or twitchings, also an unusual symptom. This symptom was also present in the second case. It is to be expected that it should sometimes occur in these severe cases of multiple neuritis in which the whole of the lower motor neurone, including the anterior horn cell, is more or less affected. The cause in this case may either have been alcohol, or an infection through the urinary tract, as he had *B. coli* pyuria, or possibly the action of the latter upon nerves previously sensitised by excessive indulgence in alcohol.

A traveller, æt. 49 years, was admitted in July, 1906, complaining of loss of power in the legs and arms. The onset was gradual, and began in the left leg a few months before; during the last month

* Allbutt and Rolleston's 'System of Medicine,' 1910, vii, p. 449.

the arms and hands had become affected. He had had no previous illness except a chill (? influenza) 12 months before. For 20 years, up to the age of 42, he had been accustomed to drink about a bottle of whisky daily in the course of his business. He said that since that age he had been a teetotaler. For some months he had been unable to get about, and had been confined to bed for over one month. He had never had syphilis.

He was well nourished, with florid complexion and dilated venules on cheeks, and watery eyes; the vessels were thickened and tortuous; the pulse full and of high tension; the apex beat was one inch outside the nipple line, the aortic second sound accentuated; the urine (sp. gr. 1010) showed no sugar or albumin; the lungs and abdominal organs appeared healthy.

There was no blue line on the gums; the tongue was tremulous but not wasted: its movements were inco-ordinated. He could not stand or sit up in bed, could not use his hands to feed himself, or hold a newspaper. When he tried to pick up anything the movements of the hand and arm had a peculiar swooping character. All movements of the hands, arms, legs, and feet were wildly ataxic in character, and this was greatly increased when his eyes were closed. Without sight he had no idea of the position of his limbs. There was no paralysis, but the ataxy practically rendered his limbs useless. Though no movements of the lower extremities were entirely absent, yet there was considerable loss of power in them, chiefly in the muscles moving the ankle and foot (periphery of limb). This was not present in the upper extremities, where the right grip measured 30 and the left 20 on inner scale of dynamometer. The muscles of arms, hands, and trunk were not wasted. On the other hand, the glutei and all muscles of lower extremities, except the ham-strings, were wasted, and the wasting was especially marked in the anterior muscles of the thigh, and tibialis anticus and peronei group. The big toes were dropped, but not the feet.

There were irregular muscular twitchings almost amounting to fibrillary tremor in the most wasted muscles. The muscles all reacted to both interrupted and constant currents, but in the wasted muscles the response to the former was diminished, and to the latter increased, without any reversal of the normal formula. The muscles were flaccid, with loss of tone: in the legs there was considerable hypotony, it being possible to put the joints into very abnormal positions without causing pain. The muscles were not tender, but there was slight tenderness over nerve trunks.

Reflexes.—Superficial: abdominal and cremasteric present, plantar doubtful. The deep reflexes were absent in both arms and legs.

Sensation to light touch (cotton-wool), pain (pin-prick), heat and cold was everywhere unaffected. Localisation of sensation was also good. The sphincters were unaffected throughout. Pupils equal, reacted to light, accommodation and consensually. Optic discs normal.

During September he had two rigors and fever, with the appearance of a quantity of pus in the urine due to a *B. coli* infection of urinary tract. The symptoms cleared up under treatment in a few weeks, but *B. coli* with a few pus cells persisted in the urine. After the first rigor there appeared partial paralysis of the right facial nerve, which gradually disappeared during the next fortnight.

The general treatment was by massage, electric baths, and systematic training in movements of the limbs. For a time the wasting of the leg muscles continued, and to such a degree that the calf muscles had the feeling described by Gowers of "a bag of worms." Fibrillary twitching in the muscles most wasted was often noticed. At the end of September he began slowly to improve, the arm muscles first recovered tone, the movements became less inco-ordinate, and he was able to use the arms and hands. Improvement in the legs followed more slowly, the fibrillary twitchings ceased, movements improved, and the wasted muscles very gradually recovered. By December he was able to stand and walk with support, and in January, 1907, with the aid of sticks.

December 20th, 1915.

Tracheal Casts in Diphtheria.—W. Fedorovsky states that expulsion of a membranous cast of the trachea after tracheotomy through the tube is not infrequent, but spontaneous expulsion through the mouth is rare. He reports a fatal case in a soldier, æt. 29 years, secondary to typhoid fever, and has collected 7 other cases from the literature, 2 in adults, æt. 19 and 33, the rest in children, æt. from 4 to 11.—*Brit. Journ. of Children's Diseases*, p. 350.

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We regret that owing to the war the number of pages must be diminished for a time.

A CLINICAL LECTURE ON SARCOMA UTERI.

By JOHN BENJAMIN HELLIER, M.D.Lond.,
Professor of Obstetrics in the University of Leeds; Hon. Obstetric Physician to the Leeds Infirmary; Examiner in Midwifery and Diseases of Women to the Conjoint Board of the R.C.S. and R.C.P., London.

THE purpose of this lecture is to discuss the subject of sarcoma uteri from the clinical side and to illustrate it with some typical cases that have been under my care in the Leeds Infirmary or in my private practice.

Sarcoma uteri is not a common disease. It is said to occur in the proportion of between 1 in 40 and 1 in 50 of cases of carcinoma uteri. In my personal experience the frequency is less. One must look for sarcoma uteri amongst cases which are supposed to be carcinoma corporis, and secondly amongst the supposed myomata, especially those which are causing pain, growing rapidly, and showing necrotic or septic changes; and also amongst soft, rapidly growing polypoid intra-uterine tumours. We may also expect that the pathologist will find sarcoma in some cases where the surgeon has suspected nothing but fibromyoma.

Sarcoma uteri is usually divided into (*a*) endometrial sarcoma, (*b*) intramural sarcoma—the *Wandsarkome* of German writers—and, rarest of all, (*c*) vesicular or grape-like sarcoma (*S. botryoides*). Examples of each are here given.

CASE 1 (see Fig. 1).—This case was at first supposed to be one of necrotic submucous myoma. It was treated by enucleation by morcellement, but recurred speedily, and when the patient returned with septic symptoms she was treated as a last resort by abdominal panhysterectomy. The uterus after removal was found to be sarcomatous. This case is interesting because it was a typical experi-

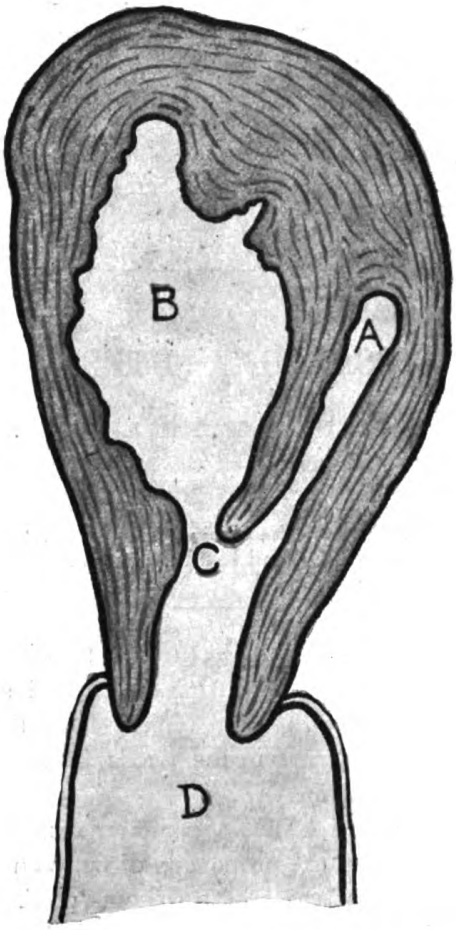

FIG. 1.—Rough sketch made after the first operation. The uterus is enlarged as a whole, and especially on the right side. A shows the normal uterine cavity; D is the vagina; B is the cavity left after enucleation of soft new growth; C is an aperture with sharp edges through which the growth was removed from B. There is a thickening of the uterine wall above B.

ence in former days when such cases were described as "recurrent fibroids" or "recurrent polypi." Probably they were all sarcomatous, either from the first or by development of sarcoma in myomata. The case occurred 14 years ago when the method of panhysterectomy by Wertheim's operation was not yet in use.

Mrs. A—, æt. 32 years, a Jewess, was admitted to the Leeds Infirmary in February, 1901. She had had no children, but two miscarriages. It was suggested that she had had a third miscarriage 10 weeks before, when some clot and necrotic tissue had been removed from the uterus, but probably such was not the case. The sound passed 4 in. into the uterus. On dilating the cervix and exploring digitally a mass of soft necrotic tissue was removed from a cavity on the right side of the normal uterine cavity, as shown in Fig. 1.

The Infirmary pathologist of that day reported that he found organised blood-clot and necrotic tissue, but did not further identify the tissue. Probably this was impossible in view of the necrotic condition of the material sent to him.

The fœtor which had previously marked the case disappeared, the hæmorrhage ceased, and the uterus became smaller, and she went home apparently well. But note the sequel. Within a month she was readmitted, the uterus was again large, and contained a large polypoid mass in a necrotic state, with foul, blood-stained discharge, and she had a high temperature. The patient was dangerously ill, but with the concurrence of my colleagues I decided that the only chance was to remove the uterus. The late Mr. T. R. Jessop saw the case with me and advised the operation. It was one of the last times that I had the advantage of his opinion. I am sorry to say that she lived only four days. The pathologist examined the uterus and found that it was a round-celled sarcoma. There were large collections of densely packed sarcoma cells with many blood-vessels with thin walls.

I will now quote a case recently under my care which has given a much better result from operative treatment.

CASE 2.—*Endometrial Sarcoma successfully treated by Abdominal Panhysterectomy.*

Miss B—, æt. 52 years, was brought to me by Dr. P. A. Steven, of Ripon, in May, 1915. She had passed the menopause 8 years before. She had enjoyed good previous health except that when about 32 she had had an illness lasting over 2 years in which she seems to have had some abdominal inflammation. Her present illness was of a year's duration, and she complained most of purulent vaginal discharge of somewhat offensive nature. She had had some uterine hæmorrhage which had twice been rather severe, but on the whole metrorrhagia had not been a very marked symptom. Since Easter she had had troublesome abdominal pain. She had lost 3 or 4 pounds in weight, and was both anæmic and sallow. There was a uterine tumour as large as a 3 months pregnancy, and this was rather tender. Dr. Steven had diagnosed malignant disease of the uterus.

On vaginal examination there was found a narrow vagina with an enlarged uterus. The os was somewhat patulous, and the tip of the finger could just reach some intra-uterine growth. There was fœtor on examination. She consented to immediate operation and came into a nursing home. Except the uterus her organs seemed to be healthy.

On May 20th I performed abdominal panhysterectomy, using the Trendelenberg position. The operation presented a curious initial difficulty. The intestines were so universally adherent that it seemed impossible to find a free abdominal cavity. The peritoneum was not thickened or reddened, but the adhesions were thin, fine, and transparent. The appendages also were adherent, but I am inclined to trace the adhesions to the former abdominal attack 20 years before.

Just above the pubes I could feel the fundus uteri covered by omental bands. With some difficulty the uterus was exposed and the appendages enucleated and the pouch of Douglas defined. After this the operation was easy. The vagina could be stripped clear as low as one desired, and the broad ligaments were not infiltrated at their base. Two Wertheim's clamps were placed on the vagina, one above the other, and the vagina was divided between them. The operation was then completed in the usual manner. I did not find any enlarged glands in the pelvis. There was very little hæmorrhage and very little shock, and she recovered without any complication, and returned home in very satisfactory condition on June 23rd.

The uterus after removal was divided by sagittal section, and is shown in Fig. 2.

Fig. 2.—Endometrial sarcoma.

Description of Specimen.

The drawing shows the uterus laid open by sagittal section. It measures 10·5 cm. externally, and 7·5 cm. internally. The antero-posterior diameter is 5 cm., and the transverse 7 cm. Below is a cuff of healthy vaginal wall 2·5 cm. long.

The uterus contains an ovoid polypoid tumour which is attached at two places to the uterine wall, that is, at its superior and posterior aspect, and also at a point on the anterior surface. It has a fairly homogeneous whitish section, but its lower end shows some necrotic change. There is a small intramural fibroid at the fundus.

Microscopic Report.

Dr. M. J. Stewart, Clinical Pathologist to the Leeds Infirmary, reported as follows: The growth varies considerably in its structure in different parts. Where it arises from the uterine wall it has the characters of a polymorphous cell sarcoma, alveolar in type, with great irregularity in size and shape of the tumour cells, and abundant mitotic figures. The vessels here are mere endothelium-lined slits in the midst of the sarcomatous tissue. In the distal (inferior) portion of the growth, the structure is largely spindle celled and benign,

with the staining characters of a leiomyoma, while the vessels are thicker walled and of a more adult type. There is a gradual transition from myomatous to sarcomatous tissue. There are numerous areas of necrosis throughout the growth. The anterior and posterior uterine walls are thinned and fibrosed, and the junction of sarcomatous and muscular tissues is very ill-defined. The free surface of the tumour is partly clothed with a thin layer of persisting corporeal endometrium, including a number of greatly stretched-out gland follicles.

It seems probable that this is an example of sarcomatous transformation of a submucous myoma.

CASE 3.—*Intramural Sarcoma* (see Fig. 3).

Mrs. C—, a Jewess, æt. 57 years, III-para, was admitted into the Leeds Infirmary in April, 1913, for a supposed myoma uteri. She was a stout and not unhealthy looking woman who had passed the menopause at 54. After 2½ years' amenorrhœa she had, in June, 1912, an attack of metrorrhagia which was followed by very little recurrence. When I first saw her there was an obvious uterine tumour reaching nearly to the navel. She had no pain, no leucorrhœa, still less any fœtid discharge, and there were no bladder symptoms. There was no doubt that the tumour was increasing in size, and I advised removal on general principles because there was a considerable uterine tumour which was growing after the menopause.

On abdominal section on April 30th, 1913, I found that there was a cyst in the right broad ligament which accounted partly for the size of the abdominal swelling. After enucleating the broad ligament cyst I performed supra-vaginal hysterectomy. The left ovary was not removed. She made an excellent

FIG. 3.—Intramural sarcoma uteri.

recovery. When the pathologist reported that the growth was malignant I removed the cervical stump by the vaginal route, and recovery was uncomplicated. She has remained well up to date.

Description of the Tumour.

Fig. 3 shows an enlarged uterus measuring 12 cm. in length, 10 cm. in antero-posterior diameter, and 7 cm. in transverse diameter. There is an interstitial tumour in the anterior wall with a fairly well-defined capsule. The section is fairly homogeneous and looks somewhat like cheese, and there is an absence of the fibrous appearance seen in myomata. At no point does the growth come through the uterine wall, which is nowhere less than $\frac{1}{2}$ cm. thick over it.

The section shows one patch of yellowish aspect as if from secondary changes. There is a small separate nodule in the posterior wall.

Pathological Report by Dr. M. J. Stewart.

The tumour is a mixed cell sarcoma, with spindle cells predominating. Numerous malignant giant cells are present, many of which have large, irregular, lobed or multiple nuclei. The growth is exceedingly cellular, and mitotic figures are present in very large numbers, not a few being multipolar or otherwise atypical. There are large areas of necrosis and of leucocytic infiltration towards the centre of the growth. At the margin the growth is fairly sharply defined, but here and there narrow strands of unstriped muscle are seen passing from the myometrium into the tumour tissue.

I really do not see how anyone was to recognise this as a sarcoma before operation. The case shows the desirability of performing total as opposed to subtotal hysterectomy in such cases.

The two forms of sarcoma hitherto described are more common in the corpus than in the cervix, but the third variety now to be mentioned is more common in the cervix, although corporeal cases are on record. I have seen but this one case of grape-like sarcoma of the uterus, and as it has already been published in the 'Journal of Obstetrics and Gynæcology of the British Empire' for August, 1914, I shall give but a brief account of it here.

Case 4.—*Sarcoma botryoides cervicis uteri* (see Fig. 4).

A 2-para, æt. 34 years, was sent to me by Dr. J. C. Lyth, of Heworth, near York, in September, 1913. She had a large polypoid grape-like tumour springing from the posterior labium of the cervix and projecting from the vulva. Her symptoms were of two years' duration, and she had twice passed portions of grape-like growth, which had been supposed to be vesicular mole. The uterus was so very mobile that after cutting through the pedicle it was exceedingly easy to remove the uterus by vaginal hysterectomy. I do not know whether abdominal hysterectomy would have given longer immunity afterwards, but at the time the

Fig. 4.—Grape-like sarcoma of cervix.

vaginal operation was so exceptionally easy that it was adopted. She made a very good immediate recovery, but there was relapse in a few months, and she died within a year from a large abdominal growth. Almost all the recorded cases have shown speedy recurrence.

DESCRIPTION OF SPECIMEN (FIG. 4) BY DR. M. J. STEWART.

Sarcoma botryoides.—This drawing shows the polypoid tumour divided from its attachment to the posterior labium uteri, but placed in apposition in its original position. The uterus above the tumour is normal to the naked eye. It measures 11 cm. in length, and the tumour 9·5 by 9·0 by 7 cm.

The surface of the growth is in large part covered by a series of grape-like processes, which arise by narrow stalks and have swollen bulbous extremities. The interior of the growth is solid.

Histologically the tumour is a myxosarcoma.

Those who seek a detailed account of sarcoma uteri should consult the article by R. Meyer and J. Veit in Veit's 'Handbuch der Gynækologie,' Bd. iii, S. 443. The principal facts about the clinical aspects of the disease are as follows:

Sarcoma uteri is mainly a disease of advancing years, being most common just after the menopause, but no age is exempt, for it is even occasionally seen in young children. The symptoms are so uncertain that it may not be possible to make a diagnosis from them alone. Sarcoma, however, grows more rapidly than myoma, and tends to cause more pain and may also cause ascites. Often it produces but little menorrhagia, and the fact that a uterine tumour is accompanied by more anæmia and sallowness than a myoma of corresponding size might be expected to produce is itself suggestive of malignancy. As the disease advances it is liable to cause metastases. These are most common in the lungs and liver, then in the intestines, omentum, and retro-peritoneal glands, but they may occur in any part of the body, including the bones. Submucous or endometrial growths bulging into the uterus or forming large soft necrotic polypi, especially when such recur after removal, are very likely to prove sarcomatous. Sarcoma uteri has sometimes produced inversion and should be reckoned amongst the causes of non-puerperal inversion. Sarcoma may cause hæmatometra and pyometra. The infiltration of the cellular tissue may be marked, but the disease does not seem to involve the ureters so frequently as carcinoma does. The disease usually runs a rapid course. About half the cases die from metastases, more than a quarter from septic processes. Other rarer causes of death are nephritis and uræmia, obstruction of the bowels, embolism, and accidental complications. The malignancy varies with the structure of the tumour. Round celled sarcoma is more malignant than cases where there is a well-marked formation of spindle cells.

The only treatment is early and complete removal. Bland-Sutton says that no uterine operation is attended by so little permanent success as hysterectomy for sarcoma.

December 27th, 1915.

The Treatment of Fracture of the Clavicle.—Couteaud has abandoned all apparatus, for he observed better results in cases treated without it. He orders rest in bed with the arm in two successive positions, which produce continuous extension. The first is the position in which the arm naturally hangs out of bed. He first recommended that this position be maintained for some days, but he has now reduced the time to an hour or hour and a half. This is a valuable modification, as the position is uncomfortable. In the second position the forearm is flexed to a right angle and rests on a stool below the level of the bed. The period has been reduced from 2 weeks to 8 or 10 days. Coaptation is perfect, and the fracture is cured in 10 days. On the third day it may not be possible to find the seat of fracture. The callus is never exuberant and the form of the bone is preserved. The clavicles become again equal, and the broken one may even lengthen to the extent of a cm. When the treatment has been adopted late—even after 2 weeks—good results are also obtained. The writer forbids massage, which may injure the callus, but after union he allows massage of the shoulder and soft parts.—*Bulletin de l'Académie de Méd.*, p. 644.

CLINICAL JOTTINGS.

(*Continued from p.* 344.)

By SAMUEL WEST, M.D., F.R.C.P.,
Consulting Physician to St. Bartholomew's Hospital.

Mouth Toilette in Severe Illness as a means of preventing many Complications.

Many of the complications of severe disease are preventable. This is obvious if we consider that they are comparatively rare in hospitals, where the nursing is good, and only common where the conditions are unsatisfactory.

Bed-sores, for example, were not at all uncommon years ago when nursing was less understood. In case of nervous disease, under the then dominant theory of trophic lesions, they were regarded as the almost natural consequence of the lowered vitality of the tissues and as almost unpreventable and incurable. Their frequent occurrence was often quoted as proof of the existence of trophic nerves —a splendid example of arguing in a circle. Not only were bed-sores common then, but patients not infrequently died of them. Now, with proper nursing, bed-sores are hardly ever seen, and if patients are admitted into hospital with them they are readily cured. The constant attention to prevent and cure them in some cases of course makes heavy demands upon the nurse; but if bed-sores develop it is generally due to want of care. The nurse should be held responsible and blamed as she deserves.

Cystitis is another case in point. This also was once regarded as more or less unavoidable, but we now know that it is generally the result of either neglect of catheterisation or of the ordinary antiseptic precautions.

The group of complications, however, to which I wish specially to refer is that which is the result of want of proper care of the mouth, for this connection is not so generally recognised. In many cases of grave illness, especially if associated with fever, the tongue is heavily coated. Most of these patients lie on the back and breathe through the mouth, with the result that the mouth and tongue become dry and caked. The air inspired brings with it germs of many kinds which grow readily under the conditions present. The fur consists of inspissated epithelium, mucus and saliva, mixed with remains of food, and teems with bacteria. The breath becomes very foul and offensive from the putrefactive decomposition which takes place, so that the condition well deserves the name given to it of sordes or filth.

Sordes has been described as if almost a necessary part of severe fever, yet it is accidental, not essential, and can and ought to be largely prevented and cured by appropriate treatment. If the air inspired passes over such foul surfaces what more likely than that it should carry infection to the lungs, and set up inflammation there—pneumonia as it is commonly called, though the infecting germ need not be the pneumococcus only. This is especially liable to occur in children where the inflammation is of the patchy multiple broncho-pneumonic type.

Some of the foul products will be swallowed and may then set up infective irritation, and thus impair appetite, cause vomiting, or excite various troubles both in the stomach and intestines, while the absorption of putrefaction products from the mouth and pharynx as well as from the gastro-intestinal tract may produce toxic symptoms which may greatly aggravate the patient's general condition.

Again the infection may spread directly to the various passages which are in connection with the mouth and pharynx. The commonest, Eustachian inflammation with otitis media as its consequence, is most likely to occur where the pharynx is gravely involved as in scarlet fever and diphtheria, but it is frequent enough in all fevers alike both in children and adults.

The posterior nares may be involved, and from thence the lacrymal duct, or the infection may extend upwards through the cribriform plate to the meninges, and excite acute meningitis, the origin of which it is sometimes so difficult to account for.

If the parotid duct be involved, swelling or even abscess of the parotid gland may result.

The curious association of parotid affections with diseases and operations of the abdomen is probably explained by the fact that in such cases the patients lie on the back, breathe through the mouth, have a very restricted diet and nothing to masticate, so that the mouth is not kept moist and cleansed by the saliva and movements of mastication. When patients are well enough to notice it, much discomfort is produced by this dryness of the mouth,

which is not relieved by the mere drinking of water. A dry powdery biscuit given to munch once or twice a day, is a great enjoyment, and goes far to remove this complaint.

All these complications may be referred to the condition of the mouth, and can all be easily and largely prevented by systematic mouth toilette. The mouth should be treated as it is in babies, washed out with a piece of rag or flannel after every feed, or, at any rate, three or four times a day, to get rid of the remains of milk or food, and afterwards rubbed all over with an antiseptic mouth-wash, containing, for instance, glycerine and borax or chlorate of potash.

Such simple precautions—that is, mouth cleanliness and disinfection—will go far to obviate many of the complications referred to. In severe illness it is as important to look as carefully to the mouth as to the back or bladder.

Rupture of the Uterus after Administration of Pituitrin.—Züllig relates the case of a woman, æt. 45 years, in her thirteenth pregnancy. The pelvis was contracted, and on each occasion it became necessary to induce labour early; even then perforation was frequently required. This time a practitioner induced labour in the thirty-sixth week. But the head was large, and the pains, in spite of administration of quinine and pituitrin, remained feeble. As there was marked contraction of the lower uterine segment, the application of forceps or version would have been dangerous. She was consequently, on July 23rd, sent to hospital with a view to Cæsarean section. The uterus was contracted, the fundus being between the umbilicus and the ensiform process. The fœtal head was below and the legs above the umbilicus. A loud uterine souffle was present, but the fœtal heart-sounds were inaudible. The pelvic measurements were: between the iliac spines, 24 cm.; between the cristæ, 29½ cm.; external conjugate, 22 cm.; diagonal conjugate, 10½ cm. (3 in.). The trochanters were on the same level, but the left iliac spine was slightly higher than the right. The pubes were abnormally deep. Under an anæsthetic an incision was made between the umbilicus and pubes. A quantity of dark blood escaped. The uterus was found to be ruptured and the fœtus and placenta were free in the abdominal cavity. They were rapidly removed and Porro's operation was performed. The child—a male—was fully developed. On August 1st a subcutaneous abscess had formed beneath the upper half of the wound and a considerable quantity of thick greenish pus was removed. The purulent secretion rapidly subsided under irrigations of peroxide of hydrogen, and on August 18th she was allowed up. Recovery ensued.

The twelfth labour, which was also induced in the thirty-sixth week, was rapidly and easily terminated. To induce labour during the thirteenth pregnancy, bougies were inserted in the uterus, and on the first day (July 22nd) 3 doses, each of 7½ gr., of quinine were administered. Labour began early on July 23rd. The os was slow in dilating, and the membranes remained intact. As the pains ceased a hypodermic injection of 0·5 c.c. (8 minims) of pituitrin was given. The pains then became stronger, and the head engaged in the brim. About 2 hours later, as the pains again ceased, the membranes were ruptured. Two and a half hours after the first or second injection of 0·5 c.c. of pituitrin was given. Strong, but not tetanic, pains followed. Fifteen minutes later contraction of the lower segment of the uterus occurred, and became so pronounced that version was excluded. The head was still firmly engaged and so presumably the rupture occurred during transport to hospital.

The writer has collected five other cases of rupture of the uterus after pituitary preparations. The chief cause is disproportion between the child and the parturient canal. In 4 of the 6 cases pelvic anomalies were present. In one case an abnormally large fœtal head was the probable cause. It has been shown that any intra-uterine manipulations, including the introduction of bougies or tents, may so damage the uterine musculature that they predispose to rupture. Such operations had taken place in 3 of the 6 cases. A multipara of 45 was certainly predisposed to rupture, but that the force of the uterine contractions was great, was shown by the extent of the laceration, which involved almost the whole of the anterior wall of the uterus from above the vaginal fornix to the fundus. The peritoneum was also torn and the placenta was completely detached. The uterus was found tetanically contracted by the side of the escaped fœtus and placenta. To this is attributable the slight loss of blood. The case again demonstrates the danger of giving pituitrin in the presence of mechanical obstruction, whether due to fœtal or maternal causes.—*Münch. Med. Woch.*, p. 743.

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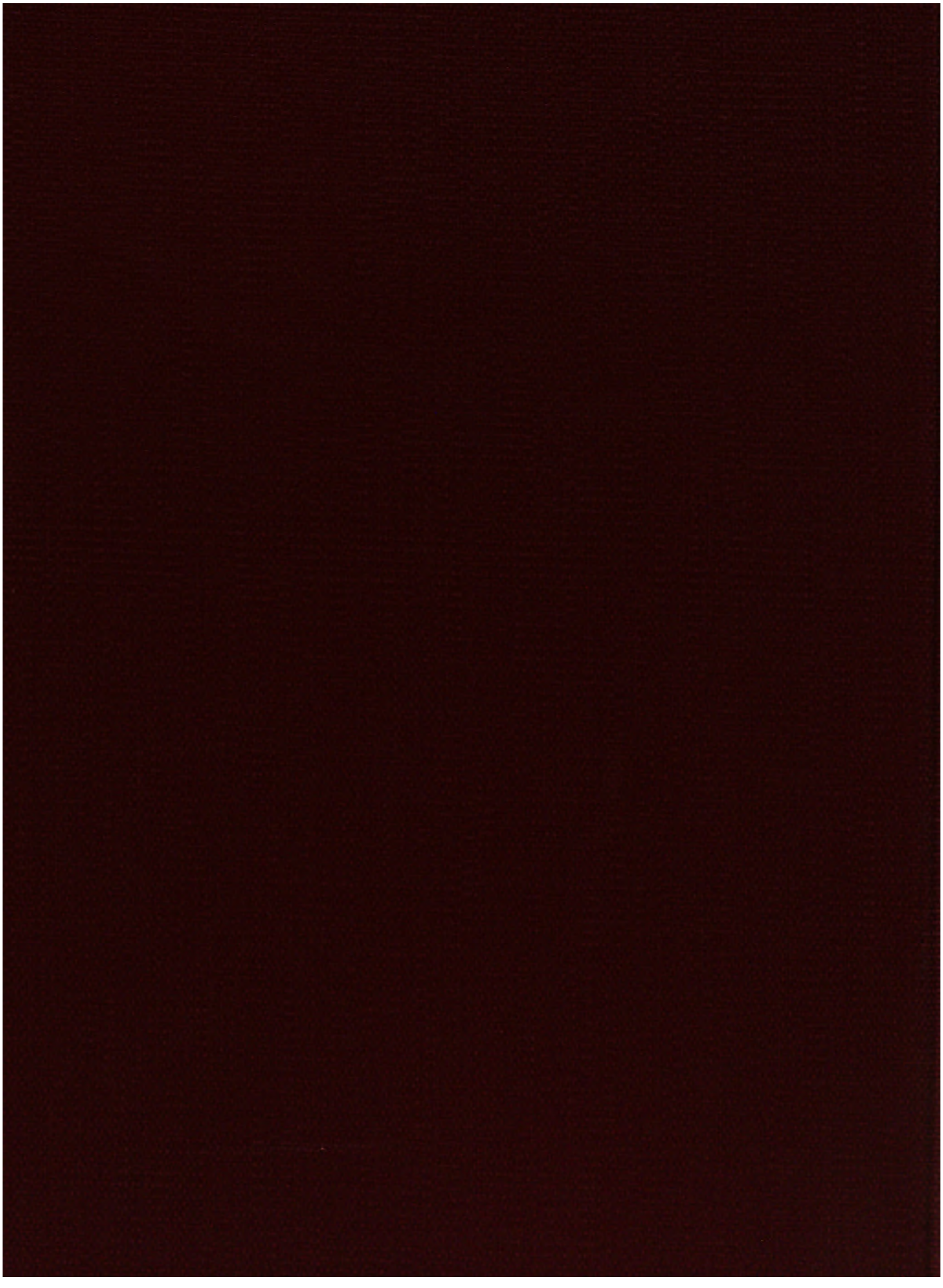
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